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Jason C. Barrett Attorney
jbarrett@eatonpeabody.com

Eaton Peabody

204 Main Street, P.O. Box 119
Ellsworth, Maine 04605
Phone 207 664 2900 Fax 207-667-3550
www.eatonpeabody.com

January 6, 2020

Via Email

Jason T. Shipp, Esq.
Goldberg Persky & White, P.C.
11 Stanwix Street, Suite 1800
Pittsburgh, PA 15222
jshipp@gpwlaw.com

Re: *Victor Coffin, an individual, and Victor Coffin, as Personal Representative of the Estate of Linda Coffin, Deceased* v. *Ametek, Inc., et al.*
Civil Action Docket No. 2:18-cv-00472-NT

Defendant Maine Central Railroad Company's Expert Witness Designation Pursuant to F.R.Civ.P. 26(a)(2)

Dear Jason:

I write to designate Victor Louis Roggli, M.D., John F. McCarthy, SC.D., C.I.H., and Stephen R. Broadhead, CIH, as experts for Defendant Maine Central Railroad Company in this case.

> **Victor Louis Roggli, M.D.** – See attached reports and materials. Dr. Roggli charges a $700 retainer fee and $700 per hour for case review, and deposition testimony. Trial testimony is charged at the rate of $600 per hour.

> **John F. McCarthy, SC.D., C.I.H.** -- See attached report and materials. Dr. McCarthy charges $450 per hour for his services.

> **Stephen R. Broadhead, CIH** -- See attached report and materials, including hearing testimony before the State of Maine Workers' Compensation Board on November 27, 2018. It is understood that you already have copies of the exhibits referenced in his workers' compensation testimony, incorporated herein by reference. Information regarding his rates will be supplemented.

Defendant reserves the right to supplement this designation, and for the identified experts to supplement their opinions and analysis, if additional relevant information comes to light through the discovery process.

All the best,

Jason C. Barrett

Enclosures
cc: Stephen Whiting, Esq., w/encl. (via email: steve@whitinglawfirm.com)

Duke University Health System
Pathology & Laboratory Services

December 12, 2019

Jason C. Barrett, Esq.
Eaton Peabody
204 Main St., P.O. Box 119
Ellsworth, ME 04605

Dear Mr. Barrett:

I have completed studies on the materials I received regarding Mr. Victor A. Coffin (Mid Coast Hospital Cytology No. NG-17-5; Maine General Medical Center Surgical No. S-17-764; PhenoPath Accession No. PP-17-1478; Duke Hospital Accession No. ML-19-289), and the findings and conclusions are summarized below.

The first specimen, labeled NG-17-5, consisted of three glass slides and two paraffin blocks prepared from a pleural fluid cytology cell block. These show atypical cells to be present singly and in small clusters.

The second specimen, labeled S-17-764, consisted of nine glass slides prepared from a right pleural biopsy. These show a biphasic malignancy involving the parietal pleura. The epithelial component consists of nests and papillary structures. Individual tumor cells are polygonal with oval nuclei, small nucleoli and moderate eosinophilic cytoplasm. The sarcomatoid component consists of anaplastic spindle cells arranged haphazardly. The tumor cells stain positive for calretinin, with the epithelial component staining strongly positive (3+) in both a nuclear and cytoplasmic distribution and the sarcomatoid component staining moderately positive (2+). The tumor cells stain positive for cytokeratins 5/6, with the epithelial component staining moderately to strongly positive (2+ to 3+) and the sarcomatoid component staining focally and moderately positive. The tumor cells stain negative for TTF-1, MOC-31 and BerEP4. Positive controls stain appropriately.

The third specimen, labeled PP-17-1478, consisted of a single glass slide prepared from the same material as the second specimen. The tumor stains positive for PDL-1 in approximately 25% of tumor cells.

The gross distribution of tumor in this case as determined by chest roentgenograms,

Duke University Health System
Pathology & Laboratory Services

computed tomography of the thorax, and direct observations of the surgeon at time of thoracoscopy, when combined with the histologic and immunohistochemical features of the tumor as described above, is diagnostic for malignant (diffuse) pleural mesothelioma, biphasic variant. (1) No lung parenchyma was sampled in this case, so I am unable to comment upon the presence or absence of asbestos bodies or asbestosis.

Thank you for referring this case for consultation.

Sincerely,

Victor L. Roggli, M.D.

Victor L. Roggli, M.D.
Professor of Pathology

REFERENCE:

1. Pavlisko EN, Sporn TA: Mesothelioma, Ch. 5, In: Pathology of Asbestos-Associated Diseases, 3rd Ed. (Oury TD, Sporn TA, Roggli VL, eds.), Springer: New York, 2014, pg. 81.

P.S. A bill for the slide review will be submitted separately. Slides and blocks returned.

DukeHealth
Department of Pathology

December 19, 2019

Jason C. Barrett, Esq.
Eaton Peabody
204 Main St., P.O. Box 119
Ellsworth, ME 04605

Dear Mr. Barrett:

You have requested my further opinion concerning the etiology of the mesothelioma in the case of Mr. Victor A. Coffin, as diagnosed in my prior report dated 12/12/19. In this regard, you have provided me with certain information pertaining to Mr. Coffin's exposure history, including excerpts from the expert report of John F. McCarthy, ScD, CIH, dated December 18, 2019.

It is my understanding that Mr. Coffin served in the US Navy from 1968 to 1971 as an aircraft electrician. He worked for the Maine Central Railroad from the summer of 1967 to December 1967 and again from 1971 to 1987. He worked as a drawbridge operator and his duties included routine maintenance and operation of the bridge. Bath Iron Works was located approximately a quarter of a mile from the Carlton Bridge. From 1988 to 2012 he worked for the US Postal Sevice as a rural letter carrier. He performed repair work on his personal vehicles from 1964 to 2003 working with brakes, clutches and engine gaskets. He also claimed exposure from home remodeling projects to joint compound, insulation, rope gaskets, roofing materials and asbestos siding. Finally, during layoffs from the railroad, he worked as a masonry tender with exposure to refractory cement and a cast-iron furnace.

Assuming that the above information is correct, then it is my opinion to a reasonable degree of medical certainty that any exposure to chrysotile asbestos from working as a bridge operator did not cause or contribute to the development of Mr. Coffin's mesothelioma. According to the expert report of John McCarthy, the upper limit of cumulative exposure to asbestos containing materials during the 17.5 year period that he worked as a Bridge Operator on the Carlton Bridge would have been on the order of 0.00958 fiber/cc-yr. These levels in my opinion are not sufficient for chrysotile to cause or contribute to the development of mesothelioma. On the other hand, exposure to commercial amphibole fibers while serving in the US Navy or performing construction work is a well-recognized cause of mesothelioma in the United States.[1,2]

Jason C. Barrett, Esq.
December 19, 2019
--page two—

Sincerely,

Victor L. Roggli, MD

Victor L. Roggli, MD
Professor of Pathology

REFERENCES:

1. Roggli VL, Sharma A, Butnor KJ, Sporn T, Vollmer, RT: Malignant Mesothelioma and Occupational Exposure to Asbestos: A clinical pathological correlation of 1445 cases. *Ultrastruct. Pathol.* **26**: 55, 2002.

2. Roggli VL, Vollmer RT: 25 years of fiber analysis: What have we learned? *Hum Pathol* **39**: 307, 2008.

CURRICULUM VITAE

Victor Louis Roggli, M.D.

Date and Place of Birth:
April 23, 1951; Winchester, Tennessee

Education:
B.A., Rice University (Biochemistry & Environmental Engineering), 1969-1973.
M.D., Baylor College of Medicine, 1973-1976.

Professional Training:
Resident, Pathology, Baylor College of Medicine and Affiliated Hospitals, July, 1976 - June 1980.

Professional Certification:
Texas State Board of Medical Examiners (FLEX), 1976.
North Carolina State Board of Medical Examiners (reciprocity), 1980
American Board of Pathology, AP/CP, 1980
American Board of Forensic Medicine, 1997

Academic Appointments;
Instructor and Chief Resident, Pathology, Baylor College of Medicine and Affiliated Hospitals, 1978-1980.
Associate, Department of Pathology, Duke University Medical Center, 7/1/80-6/30/81.
Assistant Professor, Department of Pathology, Duke University Medical Center, 7/1/81-12/31/88.
Associate Professor, Department of Pathology, Duke University Medical Center, 1/1/89-8/31/94.
Professor, Department of Pathology, Duke University Medical Center, 9/1/94-present.

Hospital Affiliations:
Durham Veteran's Administration Medical Center, Durham, N.C.
Duke University Medical Center, Durham, N.C.

Awards & Honors:
Phi Beta Kappa, Rice University
Tau Beta Pi (Honorary Engineering Society), Rice University
Phi Lambda Upsilon (Honorary Chemistry Society), Rice University
Alpha Omega Alpha, Baylor College of Medicine (1975)
Stuart A. Wallace Award (Outstanding first year medical student in Pathology), Baylor College of Medicine (1976)
Certificate of Merit for Scientific Exhibit:

1. Foster, W.L., Gimenez, E.I., Roubidoux, M.A., Sherrier, R.H., Roggli, V.L., and Shannon, R.H. The Emphysemas: Imaging-Pathologic Correlations. Roentgen Ray Society. Boston, MA, May 1991.

Cum Laude for Scientific Exhibit:

1. Foster, W.L., Gimenez, E.I., Roubidoux, M.A., Sherrier, R.H., Roggli, V. L., and Shannon, R.H. The Emphysemas: Imaging-Pathologic Correlations. Radiological Society of North America, Chicago, IL, Dec. 1991.

Educative Activities:

Junior staff position, first year medical laboratory, 1977-1979.
Instructor, Clinical Chemistry, Physician's Assistant Teaching Program, Baylor College of Medicine, 1978-1979.
Instructor, Introductory Pathology Course, Physical Therapists Teaching Program, Baylor College of Medicine, 1979.
Instructor and Senior Staff, Introductory Pathology Course, Duke University Medical Center, 1981-present.
Instructor, Systemic Pathology Course, Duke University Medical Center, 1981-present.
Instructor, Pulmonary Pathology Course, Duke University Medical Center, 1981-present.

Administrative Activities:

Research and Development Committee, VAMC (1983-1986)
Medical Records Committee, VAMC (1986-1993)
Quality Improvement Committee (1993-1995)
Director of Evening Shift, VAMC
Associate Director, EM Laboratory, VAMC (1985-1992)
Director, EM Laboratory, VAMC (1992-2006)
Member, Medical District 8 MEDIPRO Review Board (1989-1990)
Chairman, Surgical Case Review Committee (1990-2006)
Appointments, Promotions, and Tenure Committee, Duke University Medical Center (1997- 2005)
Director, National VA Electron Microscopy Program (2005-2007)

Professional Society Memberships:

International Association of Pathologists
American Association of Pathologists
College of American Pathologists
American College of Chest Physicians
American Association for the Advancement of Science
American Thoracic Society
Microbeam Analysis Society
Electron Microscopy Society of America
North Carolina Society of Pathologists
Arthur Purdy Stout Society
Society for Ultrastructural Pathology (President: 2012-2014)
International Association for the Study of Lung Cancer
Diplomate, American Board of Forensic Examiners

Research Interests:

Asbestos related diseases
Analytical transmission and scanning electron microscopy

Research Support:

Asbestos-related Diseases: Analysis of Lung Fiber Burdens. VA Merit Review 7061-001, 1985-1988, P.I. (20%)
Effects of Asbestos on Pulmonary Clearance Mechanisms. VA Merit Review 7061-001, 1988-1991, P.I. (30%)
Mechanisms of Pleural Proliferation Subsequent to Inhalation of Asbestos. VA Merit Review 7061-001, 1992-1995, P.I. (20%)
Immune Mechanisms in Human Asbestosis. NIH Grant No. 2P50 HL14212-16 (Project 3), 1986-1991, Co-Investigator (10%)
Asbestos-Induced Pleural Dysplasia in Rats, Armando E. Fraire, P.I., Victor L. Roggli, Co-Investigator, Baylor Grant BRSG 510-G-16261, 1989-1991.
Acute Lung Injury Mechanisms and Therapy, James D. Crapo, Program Project P.I.; "Core B: Pathology", Victor L. Roggli, P.I., NIH Program Project.

Invited Lectures and Seminars:

October, 1985, 51st Annual Meeting of the American College of Chest Physicians, New Orleans, LA, "Clinical Colloquium: Asbestos-Associated Diseases".
August, 1986, 21st Annual Conference of the Microbeam Analysis Society, Albuquerque, NM, "Analytical Electron Microscopy and Pulmonary Toxicology: An Overview".
April, 1988, American Society of Clinical Pathologists and College of American Pathologists Spring Meeting, Kansas City, KS, "Asbestos in the Lungs: Quantitative Approaches to the Evaluation of Lung Fibrosis and Cancer".
June, 1989, Workshop on Fiber Toxicology Research Needs, National Institute of Environmental Health Sciences, Research Triangle Park, NC, "Human Disease Consequences of Fiber Exposures: A Review of Human Lung Pathology and Fiber Burden Data".
June, 1990, The Third Wave of Asbestos Disease: Exposure to Asbestos in Place, Collegium Ramazzini, New York, NY, "Mineral Fiber Content of Lung Tissue in Patients with Environmental Exposures: Household Contacts vs. Building Occupants".
June, 1991, 11th Annual Cancer Conference and Slide Seminar, Longboat Key, FL, "Tumors and Tumor-Like Lesions of the Lung and Mediastinum".
September, 1991, American Society of Clinical Pathologists and College of American Pathologists Fall Meeting, New Orleans, LA, "Hard Metal Disease: An Enigmatic Occupational Problem".
September, 1991, Tennessee Environmental Health Association Annual Education Conference, Nashville, TN, "Asbestos and The Community".
October, 1991, 57th Annual Meeting of the American College of Chest Physicians, San Francisco, CA, "Mineral Fiber Content of Lung Tissue in Patients with Benign Asbestos-Related Pleural Disease".
April, 1993, 33rd Annual Meeting of the Houston Society of Clinical Pathologists, Houston, TX, "Malignant Mesothelioma: An Update".
May, 1993, Albany Veterans Administration Medical Center, Albany, NY, "Rarer Forms of Pneumoconiosis".
October, 1993, 59th Annual Meeting of the American College of Chest Physicians, Orlando, FL, "Mesothelioma: Pathology, Epidemiology and Pathogenesis" and "Role of Fiber Analysis in Asbestos-Associated Diseases".
November, 1993, University of Massachusetts Medical Center, Worcester, MA, "Malignant Mesothelioma: An Update of Clinical and Pathologic Aspects".
April, 1994, American Society of Clinical Pathologists and College of American Pathologists Spring Meeting, Seattle, WA, "Asbestos and the Development of Mesotheliomas".
October, 1994, Annual Scientific Meeting of the Royal College of Pathologists of Australia, Adelaide, South Australia, "Symposium on Occupational Lung Disease".
March, 1995, 5th International Conference on Environmental and Occupational Lung Disease, Miami, FL, "Clinical Methods in Occupational and Environmental Lung Disease: Pathological Classification and Reporting".
March, 1996, Society for Ultrastructural Pathology, Washington, D.C., "Mineralogic Evaluation of Lung Tissue of Persons with Malignant Mesothelioma: Is Crocidolite Asbestos the Only Type of Asbestos that Causes Mesothelioma?"
June, 1996, Occupational Pulmonary Pathology Course, São Pauulo and Fortaleza, Brazil.
August, 1996, Ultrapath VIII, Society for Ultrastructural Pathology, Oaxaca, Mexico, "Energy Dispersive X-ray Analysis and X-ray Diffraction".
January, 1997, Asbestos, Asbestosis and Cancer Workshop, Espoo, Finland.
August, 1997, 8th World Conference on Lung Cancer, Dublin, Ireland, "Tumors that Mimic Malignant Mesothelioma".
October, 1997, Mixed Dust Pneumoconiosis Workshop, Nikko, Japan.

March, 1998, ASIP Symposium for the 87thAnnual Meeting of the United States and Canadian Academy of Pathology, Boston, MA, "Molecular Genetics and Gene Therapy of Human Malignant Mesothelioma: Epidemiology and Pathology of Diffuse Malignant Mesothelioma"
October, 1998, International Academy of Pathology, Nice, France, Pleural Mesothelioma Slide Seminar (Chairman)
April 23, 1999, "Pathology of Pulmonary Neoplasms", American Thoracic Society, Post Graduate Course No. 10, Pulmonary Pathology for the Pulmonologist, San Diego, CA
April 23, 1999, "Rare Cystic and Interstitial Lung Diseases: Histopathological Pearls", American Thoracic Society, Post Graduate Course No. 3, Interstitial Lung Diseases, San Diego, CA
October, 2000, International Academy of Pathology, Nagoya, Japan, Mesothelioma and Pneumoconiosis Slide Seminar (Co-Chairman)
January 25, 2001, "Mesothelioma and Occupational Exposure to Asbestos: A Study of 1445 Cases". S. Donald Greenberg Memorial Lectures, Baylor College of Medicine, Houston, TX.
January 27, 2001, "Pathology of Pneumocomiosis." Texas Society of Pathologists Annual Mtg., San Luis Resort and Spa, Galveston, TX.
May 19, 2001, "Pathology of Pulmonary Neoplasms." America Thoracic Society, Post Graduate Course No. 22, Pulmonary Pathology for the Pulmonologist, San Francisco, CA.
February 1, 2002, "Malignant Mesothelioma: Diagnostic Challenges and Pitfalls". Grand Rounds, Georgetown Univ. Med. School, Washington, D.C.
February 23, 2002, "Scanning Electron Microscopy in Anatomic Pathology", Electron Microscopy Ad Hoc Review Group Meeting, Chicago, Il.
March 8, 2002, "Analysis of Occupational and Environmental Exposures to Dusts", International Update on Occupational and Environmental Respiratory Disease, Houston, TX.
May 17, 2002, "Pathology of Pulmonary Neoplasms", America Thoracic Society, Post Graduate Course No. 11, Pulmonary Pathology for the Pulmonologist, Atlanta, GA.
October 7, 2002, International Academy of Pathology, Amsterdam, NL, Pleural Lesions, Symposium No. 9. (Co-Chairman)
August 1, 2003, "Pneumoconiosis and Mesothelioma". Pulmonary Pathology Society Third Biennial Summer Symposium, Snowmass, CO, July 30-August 1, 2003.
October 18-19, 2003, "Respiratory Bronchiolitis Associated Interstitial Lung Disease" and "ARDS vs AIP: The Gospel According to Pratt". Symposium on Lung Disease, Baylor College of Medicine, Houston, TX.
March 6, 2004, "Asbestos-Related Non-neoplastic Lung Diseases". Pulmonary Pathology Society Companion Meeting, U.S. Can. Academy of Pathology, Vancouver, B.C.
October 11, 2004, International Academy of Pathology, Brisbane, AU, Environmental Lung and Pleural Disease, PPS Companion Meeting. (Co-Chairman)
October 14, 2004, "Asbestos Body and Asbestos Fiber Analysis in Mesothelioma Patients," International Academy of Pathology, Brisbane, AU.
February 27, 2005, "The Role of Analytical SEM in the Determination of Causation in Malignant Mesothelioma," Society for Ultrastructural Pathology, San Antonio, TX.
June 15, 2005, Pulmonary Pathology Society Fourth Biennial Summer Symposium, Lake Annecy, France, Neoplastic Lung Disease I (Co-Chairman).
April 6, 2006, "Asbestos Fiber Burden Analysis," Current Concepts in Asbestos Lung Disease, Second Annual Course, Harvard Medical School, Boston, MA.
April 7, 2006, "Pathogenesis and Pathology of Benign Asbestos Pleural Disease," Current Concepts in Asbestos Lung Disease, Second Annual Course, Harvard Medical School, Boston, MA.
May 9-10, 2006, ATSDR Expert Panel Meeting on Biomarkers of Asbestos Exposure and Disease, Atlanta, GA.
September 19, 2006, "Well Differentiated Papillary Mesothelioma," SC08 Pulmonary Pathology: Pleural Tumors, International Academy of Pathology XXVI International Congress, Montreal, Canada.

October 21, 2006, "Understanding the Terminology and Pathology of Peritoneal Mesothelioma," International Mesothelioma Interest Group, Chicago, IL
May 10, 2007, "Surgical Pathology of the Pleura," Memorial Sloan Kettering Cancer Center Surgical Pathology Course, New York, NY.
September 3, 2007, "State of the Art Mesothelioma Diagnosis: A Moving Target," Educational Session I: Mesothelioma, 12th World Conference on Lung Cancer, Seoul, South Korea.
November 2, 2007, "Twenty-five Years of Fiber Analysis: What Have We Learned?" Brigham and Women's Hospital Visiting Scholar Program, Boston, MA.
June 24-26, 2009, "Update on the PPS/CAP Guidelines for the Diagnosis of Asbestosis" and "Debate: Asbestos Exposure and Lung Cancer", Pulmonary Pathology Society Sixth Biennial Meeting, Portland, OR.
August 4, 2009, "Pleural Invasion", International Association for the Study of Lung Cancer 13th World Conference on Lung Cancer, San Francisco, CA.
August 31, 2010, "Verification of the Consensus Statement from the IMIG 2006", International Mesothelioma Interest Group, Kyoto, Japan, August 31-September 3, 2010.
January 11, 2011, "The Diagnosis of Mesothelioma in the 21st Century", Memorial Sloan Kettering Cancer Center Visiting Scholar Program, New York, NY.
February 27, 2011, "Electron Microprobe Analysis in Metal-Induced Lung Disease", Society for Ultrastructural Pathology Companion Meeting, San Antonio, TX.
August 18-20, 2011, "Update on Metal-Related Lung Disease", Pulmonary Pathology Society Seventh Biennial Meeting, New York, NY.
February 10, 2012, "The Diagnosis of Mesothelioma in the 21st Century", Brigham and Women's Hospital Visiting Scholar Program, Boston, MA.
September 14, 2012, "All Asbestos is Bad – Pro and Con", 11th International Conference of the International Mesothelioma Interest Group, Boston, MA.
June 28, 2013, "Reactive, atypical and neoplastic mesothelial proliferations: how good are our criteria?" Pulmonary Pathology Society Eighth Biennial Meeting, Grenoble, France, June 25-28, 2013.
October 30, 2013, "Pathology of mesothelioma", IASLC 15th World Conference on Lung Cancer, Sydney, Australia, October 27-30, 2013.
February 11-13, 2014, International Conference on Monitoring and Surveillance of Asbestos-Related Diseases 2014, Hanasaari Cultural Center, Espoo, Finland.
March 27, 2014, "Asbestos Exposure and Mesothelioma", Malignant Mesothelioma Diagnostic Guideline Symposium, Millennium Hilton Hotel, Seoul, Korea.
July 30, 2014, "President's Lecture: Fiber Analysis Vignettes: Electron Microscopy to the Rescue!", Ultrapath XVII, Biltmore Inn, Asheville, NC.
June 2, 2015, "Pathology and Asbestos-Related Diseases: Thresholds and Linear Models", AIHCe 2015, Salt Lake City, UT.
March 16, 2016, "Occupationally Related Pleural and Pulmonary Disorders in the 21st Century: Challenges, Pitfalls and No-nos." Short Course 59, US-Canadian Academy of Pathology Annual Meeting, Seattle, WA.
December 6, 2016, "Role of Electron Microscopy in Evaluation of Asbestos-Related Diseases Among Veterans." Department of Veterans Affairs, Electron Microscopy Program, Benchmarks for Quality in EM Webinar, Durham, NC.
March 9, 2017, "Occupationally Related Pleural and Pulmonary Disorders in the 21st Century: Challenges, Pitfalls and No-nos." Short Course 59, US-Canadian Academy of Pathology Annual Meeting, San Antonio, TX.
March 23, 2018, "Occupationally Related Pleural and Pulmonary Disorders in the 21st Century: Challenges, Pitfalls and No-nos." Short Course 59, US-Canadian Academy of Pathology Annual Meeting, Vancouver, B.C., Canada.
May 21, 2018, "Household Exposure: Correlating Cumulative Exposure (aka Dose) with Lung Digestion Analyses." AIHCe 2018, Philadelphia, PA.
June 28, 2018, "Fiber Analysis of Lung Tissue: An Overview." Ultrapath XIX, Newport, RI.
June 28, 2018, "Dimensions of Elongated Mineral Particles: A Study of More Than 570 Fibers from More Than 90 Cases with Implications for Pathogenicity and

Classification as Asbestiform vs. Cleavage Fragments." Ultrapath XIX, Newport, RI.
July 6-7, 2018, "Biphasic Mesothelioma." IASLC EURACAN Multidisciplinary Workshop on Mesothelioma Classification, Lyon, France.

Member:

U.S.-Canadian Mesothelioma Panel (April, 1987-present)
NIOSH Board of Scientific Counselors, Fiber Subcommittee (1990-present)
Credentials Committee, American College of Chest Physicians (1991-1996)
Center of Environmental and Toxicologic Pathology, American Registry of Pathology, A.F.I.P. (June, 1992-present)
International Mesothelioma Panel (1997-present)
Program Chairman, Pulmonary Pathology Society (1997-1998)
Editorial Board, Modern Pathology (July, 1997-present)
Scientific Advisory Board, Mesothelioma Applied Research Foundation (May, 1999-2010)
President, Pulmonary Pathology Society (2000-2002)
International Association for Study of Lung Cancer Pathology Panel (2004-2014)
Editorial Board, Archives of Pathology & Laboratory Medicine (February, 2005-present)
Pulmonary and Mediastinum Tumors Cancer Protocol Review Panel, CAP (September, 2006-present)
Co-Chairman, Asbestosis Committee, College of American Pathologists/Pulmonary Pathology Society (July, 2007 – present)
International Collaboration on Cancer Reporting (July, 2014 – present)
Editorial Board, Ultrastructural Pathology (July, 2015 – present)

PUBLICATIONS

Full-Length Papers:

1. Roggli, V.L.: Resident's Page, Quiz Case #2. Arch. Otolaryngol. 104:546-549, 1978.

2. Roggli, V.L., Judge, D., McGavran, M.H.: Duodenal Glucagonoma: A Case Report. Human Pathol. 10:350-353, 1979.

3. Roggli, V.L., Suzuki, M., Armstrong, D., McGavran, M.H.: Pituitary Microadenoma and Primary Lymphoma of Brain Associated with Hypothalamic Invasion. Am. J. Clin. Pathol. 71:724-727, 1979.

4. Roggli, V.L., Estrada, R., Fechner, R.E.: Thyroid Neoplasia Following Irradiation for Medulloblastoma: Report of Two Cases. Cancer 43:2232-2238, 1979.

5. Roggli V.L., Kim, H-S., Hawkins, E.: Congenital Generalized Fibromatosis with Visceral Involvement: A Case Report. Cancer 45:954-960, 1980.

6. Roggli, V.L., Greenberg, S.D., Seitzman, L.H., Hurst, G.A., Spivey, C.G., Nelson, K.G., Hieger, L.R.: Pulmonary Fibrosis, Carcinoma, and Ferruginous Body Counts in Amosite Asbestos Workers: A Study of Six Cases. Am. J. Clin. Pathol. 73:496-503, 1980.

7. Roggli, V.L., Greenberg, S.D., McLarty, J.L., Hurst, G.A., Spivey, C.G., Hieger, L.R.: Asbestos Body Content of the Larynx in Asbestos Workers: A Study of Five Cases. Arch. Otolaryngol. 106:533-535, 1980.

8. McLemore, T.L., Mace, M.L., Roggli, V.L., Marshall, M.V., Lawrence, E.C., Wilson, R.K., Martin, R.R., Brinkley, B.R., Greenberg, S.D.: Asbestos Body Phagocytosis by Human Free Alveolar Macrophages. Cancer Letters 9:85-93, 1980.

9. Mace, M.L., McLemore, T.L., Roggli, V.L., Brinkley, B.R., Greenberg, S.D.: Scanning Electron Microscopic Examination of Human Asbestos Bodies. Cancer Letters 9:95-104, 1980.

10. Roggli, V.L., Greenberg, S.D., McLarty, J.W., Hurst, G.A. Hieger, L.R., Farley, M.L., Mabry, L.C.: Comparison of Sputum and Lung Asbestos Body Counts in Former Asbestos Workers. Am. Rev. Resp. Dis. 122: 941-945, 1980.

11. Roggli, V.L., Hausner, R.J., Askew, J.B., Jr.: Alpha-1-Anti-trypsin Globules in Hepatocytes of Elderly Individuals with Liver Disease: A Report of Three Cases. Am. J. Clin. Pathol. 75: 538-542, 1981.

12. Roggli. V.L., Subach, J.A., Saleem, A.: Prognostic Factors and Treatment Effects on Survival in Erythroleukemia: A Retrospective Study of 134 Cases. Cancer 48: 1101-1105, 1981.

13. McLemore, T.L., Roggli. V.L., Marshall, M.V., Lawrence, E.C., Greenberg, S.D., Stevens, P.M.: Comparison of Phagocytosis of Uncoated vs. Coated Asbestos Fibers by Cultured Human Pulmonary Alveolar Macrophages. Chest 80: 39S-42S, 1981.

14. McLemore, T.L., Greenberg, S.D., Wilson, R.K., Buffler, P.A., Roggli, V.L., Mace, M.L.: Asbestos Associated Pulmonary Disease-An Update. Tex Med. 77: 38-45, 1981.

15. Roggli, V.L., Saleem, A.: Erythroleukemia: A Study of 15 Cases and Literature Review. Cancer 49: 101-108, 1982.

16. Roggli, V.L., Shelburne, J.D.: New Concepts in the Diagnosis of Mineral Pneumoconioses. In: Lung Pathology for the Clinician (Greenberg, S.D., ed.), Sem. Respir. Med. 4:138-148, 1982.

17. Godwin, J.D., Ravin, C.E., Roggli, V.L.: Fatal Pneumocystis Pneumonia, Cryptococcosis, and Kaposi Sarcoma in a Homosexual Man. Am. J. Roentgenol. 138:580-581, 1982.

18. Pickett, J.P., Roggli, V.L.: Rapid Histological Staining Procedures for Material from Immune-Suppressed Patients. Am. J. Med. Technol. 48:893-902, 1982.

19. Roggli V.L., McGavran, M.H., Subach, J.A., Sybers, H.D., Greenberg, S.D.: Pulmonary Asbestos Body Counts and Electron Probe Analysis of Asbestos Body Cores in Patients with Mesothelioma: A Study of 25 cases. Cancer 50:2423-2432, 1982.

20. Roggli, V.L., Pratt, P.C.: Numbers of Asbestos Bodies on Iron-Stained Tissue Sections in Relation to Asbestos Body Counts in Lung Tissue Digests. Hum. Pathol. 14:355-361, 1983.

21. Osborne, D.R., Jaszczak, R.J., Green, K., Roggli. V.L., Lischko, M., Coleman, R.E.: Detection of Pulmonary Emboli in Dogs: Comparison of Single Photon Emission Computed Tomography, Gamma Camera Imaging, and Angiography. Radiology 146:493-497, 1983.

22. Roggli, V.L., Johnston, W.W., Kaminsky, D.B.: Asbestos Bodies in Fine Needle Aspirates of Lung. Acta. Cytolog. 28:493-498, 1984.

23. Roggli, V.L., Ingram, P., Linton, R.W., Gutknecht, W.F., Mastin, P., Shelburne, J.D.: New Techniques For Imaging and Analyzing Lung Tissue. Environ. Health Persp. 56:163-183, 1984.

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1. Foster, W.L., Gimenez, E.I., Roubidoux, M.A., Sherrier, R.H., Roggli, V.L., and Shannon, R.H.: The Emphysemas: Imaging-Pathologic Correlations. Roentgen Ray Society, Boston, MA, May 1991.

2. Foster, W.L., Gimenez, E.I., Roubidoux, M.A., Sherrier, R.H., Roggli, V.L., and Shannon, R.H.: The Emphysemas: Imaging-Pathologic Correlations. Radiological Society of North America, Chicago, IL, Dec. 1991.

Dr. Roggli's 2015 thru 2019 Depositions

Date of Deposition	Patient Name	Law Firm Billed for Depo
2015		
1/6/15	Billy Prather	DeHay & Elliston
1/14/15	William Cummings	Fultz Maddox Hovious Dickens
1/21/15	James Beers	McGuire Woods
2/3/15	Larry Hoffman	K&L Gates
2/4/15	Joseph Davenport	Venable LLP
2/5/15	Linda Fishbain	Quinn Emanuel
2/9/15	Robert Gurley	Nelson Mullins Riley
2/13/15	Paul McGowan	Boyle & Brasher
2/18/15	Joyce Stockton	McGuire Woods
2/19/15	Ernest Maia	Hawkins & Parnell
2/23/15	David Greenberg	Hugo Parker
3/10/15	Judith Winkel	Quinn Emmanuel
3/13/15	Clark Collins	Mahaffy Weber
3/16/15	Kathleen Schwartz	Willcox & Savage
3/16/15	Graham Yates	Willcox & Savage
4/7/15	Norman Landry	Clements & Clements
4/8/15	Verdell Blount	Willcox & Savage
4/21/15	Ross Fordyce	Barber Law
4/28/15	Pablo Gonzalez	Willcox & Savage
5/5/15	Walter Koepper	Parler Wobber

5/21/15	Narcisco DeLeon	Crimando & Cleland
5/26/15	Eric Ross Phillips	Leath Bouch Law
6/10/15	Joseph DeLise	DeHay & Elliston
6/30/15	Elijah Sparkman	Nelson Mullins
7/7/15	David Baird	DeHay & Elliston
7/22/15	Gavin Petillo	Thompson Bowie
7/27/15	Rita Treutel	Forman Perry Watkins Krutz
7/28/15	Vernon Turner	Unglesby Law
8/4/15	Richard Gilb	Willcox & Savage
8/11/15	Garland Pepper	O'Connell Tivin Miller Burns
8/13/15	Danny Maxwell	McGuire Woods
8/24/15	Larry Lee	Alston Bird
9/2/15	Joe Trejo	Bassi Edlin Hughie & Blum
9/3/15	Maurice Shrum	McGuire Woods
9/8/15	James DeSouza	Segal McCambridge Singer
9/9/15	Thomas Dandridge	Nelson Mullins Riley
9/10/15	Willard Krumweide	Segal McCambridge Singer
9/10/15	Arthur Kroeger	Lankford Crawford Moreno
9/11/15	Kenneth Hotard	Martzell & Bickford
9/14/15	Stewart Poore	McGuire Woods
9/16/15	Richard Roop	McGuire Woods
9/17/15	Richard Russell	Rasmussen Willis Dickie
9/24/15	Martha Faye Joseph	Foley & Mansfield
10/8/15	Cami Bivert	Schiff Hardin
10/12/15	Johnny Penegar	Goodman McGuffey Linsey

10/20/15	Richard Roop (continuation)	McGuire Woods
10/20/15	Michelle Zierer	McGuire Woods
10/21/15	Michelle Zierer (continuation)	McGuire Woods
10/27/15	Jesus Vega	Dentons LLP
10/28/15	Virginia Schiszler	Berkes Crane Robinson
11/11/15	Paul McCulley	DeHay & Elliston
12/02/15	Irma Saldana	DeHay & Elliston
12/3/15	Francisco Urcuyo	DeHay & Elliston
12/7/15	Kenneth Reed	Willcox & Savage
12/14/15	Victor Jasniy	DeHay & Elliston

2016

1/6/16	Jesus Vega – Continued	Dentons LLP
1/21/16	John Dickerson	Evert Weatersby Houff
2/29/16	Charles Smith	Archer Norris
3/8/16	Terry Poe	Berkes Crane Robinson
3/29/16	Michael Talbot	Martzell & Bickford
4/21/16	James Hutto	Nelson Mullins Riley
5/2/16	Ronald Hill	Waters & Kraus
5/4/16	Billy Parrott	Smith Anderson Blount
5/5/16	Rosie Trusclair	Plauche Maselli Parkerson
5/16/16	Gary Scott Smith	Waters & Kraus
6/2/16	James Angus	O'Connell Tivin Miller Burns
6/17/16	Leonard Smith	Willcox & Savage
6/14/16	Gerald Hake	Selman Law Office

7/21/16	Louis Lowell	Gordon & Rees
7/28/16	Thomas Gatten	McGuire Woods
8/1/16	Clifford Johnson	Baird & Baird
8/1/16	Randy Napier	Baird & Baird
8/2/16	Robert LaDuca	The Powers Law Firm
8/3/16	Hayward Theriot	Walters Papillion
8/11/16	Theresa Gaubert	Kean Miller
8/15/16	Abraham Castillo	O'Connell Tivin Miller Burns
8/29/16	Thelma Oster	Bice Cole
8/31/16	Dennis Britt	DLA Piper
9/14/16	Nolan Lamb	Selman Breitman
9/26/16	Frederick Hensley	Nelson Mullins Riley
9/27/16	James Wagner	Hawkins & Parnell
9/29/16	Charles Smith	Archer Norris
10/6/16	Anna Blount	Selman Breitman
10/24/16	Donald Moody	Selman Breitman
12/7/16	Lewis Pyatt	Haynsworth Sinkler Boyd
12/8/16	Otto King	Plauche Maselli Parkerson
12/14/16	Ralph Pennington	Willcox & Savage

2017

2/1/17	Melissa Sweet	Willcox & Savage
2/3/17	Jose DeLaO	Willcox & Savage
2/15/17	John Mehalick	Willcox & Savage
2/17/17	Raymond Parker	Leader & Berkon

2/20/17	Lynda Berry	K&L Gates
2/27/17	James Smith	Boyle Brasher
3/20/17	Larry Watson	Selman Law
4/10/17	Bobby Farmer	Evert Weathersby
4/17/17	Edward Williamson	Rumberger Kirk & Caldwell
5/15/17	James Smith (2nd depo)	Boyle Brasher
5/24/17	Ronald Carroll	O'Connell Tivin Miller Burns
6/1/17	Charles Kenyon	Foley & Mansfield
6/2/17	Frank Hart	Foley & Mansfield
6/26/17	Jacob Burg Adams	Plauche Maselli Parkerson
6/28/17	Steven Holtsclaw	Manion Gaynor Manning
7/6/17	Wade Gore	O'Connell Tivin Miller Burns
7/10/17	Loretta Ney	Riley Safer Holmes Cancila
7/18/17	Brent Menard	Manion Gaynor Manning
7/25/17	Paul Casterline	Lankford Crawford Moreno
8/28/17	Clifton Younce	Willcox & Savage
8/29/17	Jesse Gonzales	Hassard Bonnington
9/6/17	Gerald Kessinger	Riley Safer Holmes Cancila
9/18/17	Velma Searcy	Willcox & Savage
9/25/17	Roger Williams	Lankford Crawford Moreno
10/16/17	Sharron Smith	Imai Tadlock Keeney
10/24/17	Jerry Bagwell	Forman Watkins & Krutz
11/22/17	George Williams	Tybout Redfearn & Pell
12/4/17	Clyde Crowe	Goldberg Segalla
12/13/17	Vicki Williams	Stoll Keenan Odgen

2018

1/9/18	Joseph Gonzon	Tybout Redfearn & Pell
1/10/18	Helene Kohr	Hughes Hubbard & Reed
1/16/18	Rachel McCrary	Wilson Smith Cochran
1/30/18	Robert Rash	Roven Kaplan
2/8/18	Enrique Dominguez	Archer Norris
3/5/18	Richard Berg	Hughes Hubbard & Reed
3/12/18	Michael Donohue	O'Connell Tivin Miller & Burns
3/15/18	William Hawkins	Willcox & Savage
3/28/18	William Choate	Lewis Brisbois
4/11/18	James Bookout	Boyle Brasher
4/13/18	Thomas Bardusk	Foley & Mansfield
4/19/18	Lloyd Bailey	Hassard Bonnington
4/25/18	Donald Ward	Sinars Rollins
5/23/18	Wayne Bourgeois, Sr.	Martzell & Bickford
6/12/18	Patrick Jack	Gardner Trabolsi & Associates
6/14/18	Jerry Crawford	Nelson Mullins
6/19/18	Donald Knutson	Gordon & Rees
8/20/18	Louis Summerlin	Cetrulo LLP
8/27/18	Alcee Anderson	Nelson Mullins
9/6/18	Steven Bolin	Willcox & Savage
9/18/18	Benny Anthony	Pessin Katz Law
9/19/18	Jeffrey Watts	Foley & Mansfield
10/1/18	Michelle Price	Hubbard Mitchell
10/2/18	Alfred Butchko	Gordon & Rees

10/8/18	Leslie McGrew	Bienvenu Bonnecaze Foco
10/16/18	Dianne Edwards	Maron Marvel Bradley
10/23/18	Earl Gisclair	Ron Austin Law
10/24/18	James Thomas	Sinars Rollins
11/1/18	Robert Stewart	Willcox & Savage
12/10/18	Alex Williams	Gordon & Rees
12/11/18	Brian Brown	Schnader Harrison Segal
12/12/18	Kenneth Hendrix	Barber Law Firm
12/20/18	Sarah Krentz	Hunton Andrews Kurth
2019		
1/30/19	Stephen Fowlkes	Willcox & Savage
2/13/19	Charles Bourg	Galante & Bivalacqua
3/11/19	Jody Ratcliff	Kaleo Legal
3/28/19	Rodolfo Gutierrez	Forman Watkins
4/1/19	Patricia Schmitz	Foley & Mansfield
4/2/19	Beatrice Bourque	Nelson Mullins
4/17/19	Owen Gabriel	Bullivant Houser Bailey
4/18/19	Eric Sinor	Foley & Mansfield
5/13/19	Owens Edge	Jones Walker LLP
5/20/19	Robin Gould	Frilot LLP
6/4/19	Thomas Florence	Kaleo Legal
6/5/19	John Glenn	Nelson Mullins
6/11/19	Barbara Reed	Hendler Lyons
6/12/19	Jeremy Smith	Motley Rice
6/17/19	George Crudge	Gordon & Rees

6/19/19	George Crudge (continuation)	Gordon & Rees
7/9/19	Harold Hilliman	Dentons
7/17/19	Ronald Smith	Willcox & Savage
7/30/19	Richard McClendon	Willcox & Savage
7/31/19	Elray Lege	Baggett McCall Gaughan
8/1/19	Alfred Bennett	Kaleo Legal
8/6/19	David Phelps	Boyle Brasher
8/27/19	Terry Steinberg	Swanson Martin & Bell
8/29/19	Sharon Barrilleaux	Frilot LLC
9/4/19	Amos Webb	Willcox & Savage
9/10/19	Melody Lewis	Swanson Martin & Bell
9/26/19	Sarah Krentz	Foley & Lardner
10/8/19	Charles Fuller	Willcox & Savage
10/10/19	Peggy Posey	Willcox & Savage
10/14/19	Rudie Klopman-Baerselman	Bullivant Houser Bailey
10/17/19	Gordon Andersen	Foley & Mansfield
10/22/19	Jeremiah Hartle	Browning Kaleczyc Berry Hoven
10/23/19	Carolyn Ward-Wiman	Moran Reeves Conn
10/24/19	David Lara	Benjamin Law Group
10/28/19	James McAllister	Hubbard Mitchell Williams
11/6/19	Jean Roy	Hugo Parker
11/11/19	Russell Mahoney	Foley & Mansfield
11/14/19	Gordon Andersen	Foley & Mansfield
11/25/19	Larry Woolard	Pinto Coates Kyre & Bowers
11/26/19	Suzanne Garon	Boyle Brasher

12/05/19	Calvin Harris	Forman Watkins Krutz
12/12/19	Carolyn Ward-Wiman (continuation)	Moran Reeves Conn
12/16/19	Thomas Gaskill	Nelson Mullins Riley
12/17/19	Kris Elizarraras	Foley & Mansfield
1/2/20	Forrest Lewis, Jr.	Willcox & Savage
1/6/20	Deborah Creech	Forman Watkins

TRIAL TESTIMONY

PLAINTIFFS

James Washington (MN)	Charles Arnold (TN)	Roy Boyette (TX)
Manuel Carvalho (HI)	Daniel Johnstone - I (LA)	Steven Douglas (TX)
Ewing B. Edwards (TN)	Ed Lindley (NC)	Gerald England (MD)
Frank Smith (RI)	Clarence Stevens (CA)	William Duckett (TX)
James O. Bullard (FL)	Ross Grossnickle (MD)	Nicholas Baione (FL)
Gregory Davenport (VA)	Martin Mason (TX)	Milton Jenkins (CA)
Bernard (WA)	Kenneth Thrapp (TX)	Edward Norris (FL)
Roger Shack (AL)	Robert Yeager (TX)	Barnes and Wasiak (TX)
John E. Richardson (FL)	Charles Hughes (LA)	Benjamin Baker (WV)
Leonard Mays (FL)	Damon Quick (WV)	Victor Hellquist (CA)
Ralph Turley (WV)	Daniel Johnstone - II (LA)	John Aday (AL)
Garvin Boiter (SC)	Robert Lofton (TX)	Carl Golightly (KY)
William Drake (NC)	Robert L. Verdin (MD)	Charles Allen (FL)
Cynthia Archer (FL)	Roy Boyette (LA)	Philip C. Skleros (NJ)
Cimino et. al. (TX)	Harry DeLong (WV)	Connell and Parkinson (FL)
Daniel Boyce (TX)	Nicholas Haluskey (CA)	Lillie Irene Permuy (FL)
Peters and Jones (WV)	Benny Gerald Horne (NC)	George E. Prekler (NC)
Sego and McElhaney (FL)	Arthur Carter (DE)	Robert L. Stone (TX)
Mickens, Taylor, Byford, McBryde and Gray (TX)		Dean-Steffens (FL)

TRIAL TESTIMONY

PLAINTIFFS (cont.)

William E. Hohman (MD)	Roy Shaw (WA)	Leon Swindler (LA)
James Blanchard (KY)	Herman Parks (VA)	Jean Connor (FL)
Rita Mae Schmidt (TX)	Whatley (TX)	O'Flynn (LA)
Samuel Culverhouse (NC)	Piper, McFadden, Fertig (MD)	Goodwin (TN)
William V. Monahan (CA)	Paul Cochran (TX)	Ben Baker (TX)
Orval Vinson (AL)	John Roth (KS)	Johnny Butler (GA)
Carl Golightly II (KY)	Patrick Henry Clark (LA)	J. Karbiwnyk (FL)
Knoch & Mueller (MD)	Christine C. Boudoin (LA)	Carlton Crane II (FL)
Cleo Elmore (NC)	Edward Snoozy (FL)	P. McCormick (DC)
Lassiter & Morgan (FL)	Stanley Lesnick (MD)	Russell Meinert (TX)
Carlton Crane (FL)	Robert Gillespie (WV)	Thomas Hadley (IN)
Mizell - Durant (FL)	Luther Wilkerson (TX)	WV Mass Trial (WV)
James Gardiner (TX)	Ridenour-Eitemiller (MD)	Mildred Wiley (IN)
James V. Redd (FL)	Svoboda-Siedlecki (MD)	Samuel Wheat (TX)
Charles White (TX)	A.B. Johnson (FL)	Earl E. Brown (TX)
Edmund Cooper (DC)	Eastburn-Arthur (DE)	Charles Bendit (CA)
James Cecil Chaney (TX)	John Break (MI)	Leo Woodburn (FL)
LaVerne Ruff (CA)	Otto Kelley (WA)	Charles Green (TX)
Zumas-Balonis (MD)	Charles Reiff (TX)	Austin Clement (LA)

TRIAL TESTIMONY

PLAINTIFFS (cont.)

Raymond Denkeler (TX)	John Shelton (TX)	Joseph Torrejon (LA)
Howard Aylor (TX)	Martin Little (SC)	Katie Carter (PA)
Richard Callahan (OH)	Douglas Faulkner (LA)	Thomas Madden (TX)
Freeman Gilbert (TX)	Raymond Peterson (FL)	Thomas Pipich (CA)
Leo Spiegelhauer (TX)	Billy Ray Meadows (TX)	Linda Welch (MO)
Joaquin Banda (TX)	Constance Mehlman (NJ)	Gail Routh (FL)
Jose Rubio (TX)	Victor Brunell (LA)	Schoonover (WI)
Burl Butler (MS)	Joseph Breaux (TX)	Maurice Flood (IL)
Frank Goodman (GA)	Harvey Harrison (TX)	Beverly Davis (FL)
Alvin Hebert (LA)	Elmo Zumwalt (TX)	Samuel Dryden (TX)
James Salierno (TX)	Fred Wakeland (IL)	Dante DeJohn (IL)
Larry Hollis (DE)	Paul Dauzy (MI)	Boyce Innerarity (LA)
Kenneth Fowler (IL)	Robert Robinson (OH)	Joseph Breaux (LA)
Robert Looman (TX)	Rose Wiener (PA)	Samuel Heron (LA)
R. Bailey-D. Smith (TX)	Webb & Wallace (WV)	William Lamb (GA)
Suzanne Jones (FL)	Kenneth Zimko (LA)	Lawrence Berry (TX)
Jerrold W. Anderson (WI)	Billy Roberts (IN)	Walter Graves (LA)
Barbara Hoffman (GA)	Willie T. Moore (TX)	Ms. Behringer (TX)

TRIAL TESTIMONY

PLAINTIFFS (cont.)

Bill Carney (AR)

Leila Schwartz (FL)

Sam Wallace (TX)

Jimmie Lang (LA)

James Terrance (LA)

Bruce Spillman (LA)

Ray Rando (LA)

Jack Pounds (CA)

Richard Belt (CA)

James H. Bennett (IL)

Elreece McKinney (TX)

Lambert Winnemueller (WI)

Joseph Ferlanti (FL)

Eileen Honer (CA)

Leslie Sabin (UK)

Leroy Conway (MD)

Betty Lou Bello (LA)

Jay Morse (FL)

Daniel Begley (FL)

Tommy White (LA)

Myra Williams (LA)

Elray Lege (LA)

TRIAL TESTIMONY

DEFENDANTS

Wilmer Nall (IL)	Michael Field (OR)	William Fraysure (OH)
Danny Southers (FL)	Paul Caruolo (NY)	Paul Kubik (OH)
Kaowili - I (HI)	Walters and Tyson (LA)	Chris Hilsenbeck (WA)
Kaowili - II (HI)	Lisa Pransky (MD)	Michael Sparks (TX)
Joseph Lotz (LA)	Mark Lewis (CA)	Eldon Perkins (NY)
John Kuiper (NJ)	Renee Prior (CA)	Carl Saville (MD)
Lerch vs. Northwestern (IL)	Michael Missik (OH)	Chuck Norris (KY)
CCR Settlement Hearings (PA)	Linda Batten (TX)	Rolf Lindstrom (OH)
Ernest Tryon (TX)	James Henderson (SC)	Noah Pride (NY)
Ernest Tryon (TX)	David Taylor (CA)	Bernard Mayer (NY)
Rockwool (LA)	Polly Burns (CA)	Earl B. Coxen (OR)
Douglas vs. Villines (NC)	Robert Jones (GA)	Julian Petruzzi (IN)
Ethel Marie Granski (MD)	John Matteson (NY)	Clarence Bentley (FL)
Abernathy (TX)	Douglas McCarthy (CA)	Charles Porter (OH)
John Cardinal (NY)	Paul Wilson (MD)	Ernest Coulter (WA)
William Perate (PA)	WV Mass Trial (WV)	George Risk (PA)
Brown, Zorn & Cone (GA)	Douglas Anderson (CA)	Charles Padilla (CA)
Braxton Colley (MO)	Charles Flowers (GA)	George Bullen (NY)

TRIAL TESTIMONY

DEFENDANTS (cont.)

Garland Jones (VA)	Larry D. Smith (MS)	Roy Knight (GA)
Edward C. Martin (NY)	Willie R. Martin (CA)	Norman Shoopman (IL)
Flora Franklin (KY)	Robert Smith (CA)	William Harrell (CA)
James L. King (VA)	Clark Kirkland (TX)	Nasseem Farag (CA)
Joel Rosenberg (NY)	Richard Simpson (NY)	Gordon Bankhead (CA)
Joyce Windnagle (FL)	Ronald Drabczyk (NY)	Chester Morrison (CA)
George T. Dancho (IL)	Henry Barabin (WA)	James D. Lokey (VA)
MaryAnn Ormonde (CA)	Richard Adams (CA)	Richard Steiner (CA)
Carl Saville (MD)	Jimmy Toole (GA)	Charles Garrison (FL)
Leonard Shafer (NY)	James Sweeney (PA)	Haskell Stillman (CA)
Robert Donlan (MA)	Fred Rich (FL)	Anna Evans (GA)
George T. Dancho II (IL)	Milton Ferrell (FL)	Lloyd Benton (NY)
Lynda L. Daly (FL)	Larry Williams (IL)	Luis Torres (FL)
Randy Elson (IL)	Robert Hardick (VA)	Carolyn Esters (CA)
Gerald Bretzke (MN)	George Clark (CA)	John Bristow (VA)
John Picinic (NJ)	Vickie Warren (UT)	Armin Thoma (NJ)
Lynda L. Daly (FL)	Susan Schrader (NJ)	Johnnie Jones (NC)
James L. King (VA)	William Aubin (FL)	Marline Petitpas (CA)

TRIAL TESTIMONY

DEFENDANTS (cont.)

Michael Galliher (DE)

Daniel Skinner (CA)

Glenda Vega (NY)

Julius Sanders (FL)

Richard Delisle (FL)

Lloyd Garvin (SC)

James D. Lokey (VA)

Reydel Muniz (FL)

Herbert Goins (GA)

Willie Robinson (OH)

Joseph Salerno (PA)

Gary Hampton (FL)

Charlie Stampley (MS)

Joseph Muldoon (SC)

Michael Galier (OK)

Pablo Gonzalez (FL)

Joyce Stockton (TN)

Sherry English (PA)

James Harkin (CA)

Dennis Seay (SC)

William Cummings (KY)

Victor Jasniy (CA)

Michael Argento (NJ)

Dennis Britt (FL)

James Angus (CA)

Paul McCulley (IA)

Joseph Bowers (PA)

Robert Sprague (OR)

Velma Searcy (CA)

Donald Knutson (CA)

Louis Summerlin (MA)

Jerry Bagwell (LA)

Robert Stewart (DE)

Willis Edenfield (NJ)

Stephen Fowlkes (VA)

Richard LeFrak (NY)

Anita Creutzberger (NJ)

Rodolfo Gutierrez (NM)

Alfred Bennett (MO)

Ronald Smith (CA)

Robert Calvey (OH)

Helen Schwartz (MO)

Environmental Health & Engineering, Inc.
180 Wells Avenue, Suite 200
Newton, MA 02459
TEL 800-825-5343
781-247-4300
FAX 781-247-4305
www.eheinc.com

Victor Coffin

Plaintiff
v.
Ametek Inc., et al
Defendants

REPORT OF JOHN F. MCCARTHY, SC.D., C.I.H.

I, John F. McCarthy, declare as follows:

1. I am President of Environmental Health & Engineering, Inc. ("EH&E"), with more than thirty years of experience in environmental exposure assessment. My particular focus has been on multimedia (air, water, soil) source characterization and assessment that could result in human exposure to contaminants in community, industrial and non-industrial settings. My work has involved the assessment and characterization of exposures to particles and gases, including respirable particulate matter and volatile organic compounds, among others. I have been employed by EH&E since January of 1988. Prior to that, from 1978 to 1988, I was employed as a research scientist by the Massachusetts Institute of Technology Energy Laboratory where I studied particulate and vapor production from various high temperature processes and evaluated health impacts of resulting airborne contaminants. My work has involved design and implementation of field and controlled chamber studies to determine the source, transport, and fate of various airborne contaminants, as well as evaluating their possible exposure potential and risk.

2. I am certified by the American Board of Industrial Hygiene in Comprehensive Practice. I received my Master of Science degree in Environmental Health Sciences, specializing in Air Pollution Control and Industrial Hygiene, from Harvard University in 1978. I received my Doctor of Science degree in Environmental Science and Physiology, specializing in Physical Science and Engineering, from Harvard University in 1987. I have published numerous scientific papers on air quality analysis, including exposure characterization in indoor environments, as well and served as the Program Director for a number of research projects for the U.S. Environmental Protection Agency's (EPA) Indoor Environmental Quality Division and the National Institute of Occupational Safety and Health's Division of Respiratory Disease Studies. I have served on several private, governmental and professional organizations' health and safety committees. I was the Principal Investigator on the research studies related to problem drywall conducted by EH&E, on behalf of the Consumer Product Safety Commission. My qualifications and experience are further detailed in my curriculum vitae, which is attached hereto as Appendix A.

3. I have been asked to review a number of documents relevant to the potential for occupational exposure to asbestos fibers in the air while Mr. Coffin worked as a Bridge Operator for the Maine Central Railroad Company (MCR) and the Maine Department of Transportation (MDOT) at the Carlton Bridge, situated in Bath, Maine. I have also reviewed various articles and reports in the general literature, and documents specifically related to Mr. Coffin, and have been asked to render an opinion as to whether Maine Central Railroad:

a) Contributed to an unsafe environment for him during his normal course of activities, that he stated he undertook and experienced as a Bridge Operator.
b) Contributed to potential levels of asbestos-containing particles, in the areas in which he was present, that were considered excessive and the potential hazards were foreseeable.

4. I reviewed a variety of relevant case materials including:

- The Complaint filed November 15, 2018
- Deposition of Mr. Victor Coffin, dated September 17 and 18, 2018
- Report by Northeast Test Consultants titled Professional Review for Asbestos Exposure relating to Victor A. Coffin v. State of Maine/Dept of Transportation, dated August 17, 2018.
- Transcript of Stephen Broadhead at Worker's Compensation Hearing, November 27, 2018.
- URS Corporation's Asbestos & Lead-Based Paint Compliance Audit Report from March 2005 for Brunswick Post Office.
- Cardno ATC's Limited Asbestos-Containing Material and Lead Containing Paint Inspection Report from July 2013, for Brunswick Post Office.
- Additional documents are detailed in Appendix B

BACKGROUND

5. Mr. Victor Coffin was born on November 26, 1948 in Bath, Maine.[1] He graduated from Brunswick High School in 1967.[2] He has four children: Victor, Nathan, Jonathan and Ryan.[3] He was diagnosed with Mesothelioma on January 18, 2017.[4]

[1] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 11, 20-21
[2] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 41-42, 70-71; Exhibit 2
[3] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 12
[4] Complaint filed November 15, 2018; Victor Coffin v. Ametek, Inc., et al.

Relevant Chronology of Mr. Victor Coffin's Work History

- Summer 1967-December 1967 Maine Central Railroad (Assistant Drawbridge Operator on the Carlton Bridge for approximately six months) [5]
- 1968-1971 Navy (Aircraft electrician) [6]
- 1971-1987 Main Central Railroad (MCR) (Worked where boxcars were rebuilt and maintained for about a month with remaining time spent as Drawbridge Operator on Carlton Bridge) [7]
- 1987-1988 Maine Department of Transportation (Chief Operator of Carlton Bridge for a 10-month timeframe) [8]
- 1988-2012 U.S. Postal Service (Rural letter carrier) [9]

6. Additionally, Mr. Coffin reported potential dust exposure scenarios during various activities that he would undertake, including home improvement at the family home while growing up, during personally building his own home, home automotive work, and work as a Masonry Tender for supplemental income.

United States Navy

7. After a 12-14 week boot camp in 1968, Mr. Coffin was sent to Jacksonville, Florida for aircraft electrician training for six months; training involved 90 percent classroom instruction and 10 percent hands-on work on obsolete Korean war aircraft, working on such things as fuel gauges and lights.[10] After finishing training, he was sent to his permanent duty station in Cecil Field, Florida until he went to Vietnam in 1970.[11] He received classroom training on the Navy A-1 Corsair during this time involving navigation system components and terrain guidance system components among other general training; he was an aircraft electrician during this time.[12] As an aircraft electrician, he was responsible for maintaining a squadron of Corsairs, about 20; he worked on all the electrical components except for the basic radio and radar, including all the external lights and all of the gauges and switches, such as landing gear switches.[13] He also reported that he may have worked with aircraft components containing gaskets such as during replacement of sensors on engines.[14]

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[5] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 41-42, 70-71; Exhibit 2
[6] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 124-132
[7] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 77-78
[8] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 160
[9] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 160-161
[10] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 124-127
[11] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 128, 134
[12] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 129-132
[13] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 135-136
[14] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 138-139

8. While in the U.S. Navy, he was also assigned to a temporary additional duty at a Marine Corps air station in Yuma, Arizona for about seven months after Cecil Field; his duties stayed the same and continued to involve performing maintenance on Corsairs.[15] He went back to Cecil Field after Arizona, since it was a temporary assignment, and was then stationed at an army base in Eustis, Virginia for about a month to get training on the electrical systems on helicopters.[16] On August 31, 1970, he was sent to Vietnam to work at a helicopter base as an aircraft electrician.[17]

9. At the helicopter base in Vietnam, there were about 20 Bell Huey helicopters he would work on.[18] He worked on various electrical components such as the compass, all the lighting, and all the wiring within the helicopter.[19] The only time he felt he would have worked on anything containing asbestos during this time would be when he would have had to help mechanics change an engine which contained gaskets; he did not report how often he would have done this.[20] He characterized this as minimal involvement work which required helping lift up engines for replacement, he was not involved in taking apart engines.[21] Mr. Coffin reported that when he was in Vietnam and working on the Hueys, electricians would be working alongside mechanics and there could be multiple different categories of maintenance people working at the same time on the helicopters.[22]

10. Mr. Coffin recalled an asbestos shield being present in the crew compartment between where they sat and the engine – the fuel tank was directly behind them, "there was an entire wall of - - it looked like a silver quilt, and it was asbestos, and it just kept the crew component away from fuel if that ever happened".[23] "This shield was to keep the fire, if there was a crash, away from the pilot; he was told this was asbestos".[24] "The shield was about 8-9 feet wide, 5 feet high, a quarter to half inch thick and was attached to the back wall of the helicopter".[25] "There was diamond stitching running through this silvery fabric".[26] He recalled that some of the shields had bullet holes and shrapnel holes.[27]

11. He also recalled a mitt/asbestos pads that were used by pilots to remove the hot gun barrel from the helicopters– although he did not use these, he reported having to move these "asbestos pads" once in a while if they had to get to something.[28] He never wore a respirator during

[15] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 145
[16] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 146, 149
[17] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 128, 134, 146
[18] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 147
[19] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 149-150
[20] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 150
[21] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 435
[22] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 517-518
[23] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 150-151, 153
[24] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 153
[25] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 428-429
[26] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 429
[27] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 430
[28] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 151, 153, 432

any of his aircraft work.[29] He recalled that he had an accident while working, which required surgery on his spine, in which he was hospitalized for 30 days while in Vietnam after which he went back to his regular duties.[30]

Maine Central Railroad (MCR) Summer of 1967–December 1967, and 1971–September 1987

12. Mr. Coffin started working at MCR in the Summer of 1967 and worked for about six months until December 1967 before joining the Navy; he worked as an Assistant Drawbridge Operator in the Signal Department and was assigned to work on the Carlton Bridge, located between Bath and Woolwich, Maine; he worked 11 a.m. to 7 p.m.[31] His job involved routine maintenance and operation of the bridge, which included, greasing, maintenance of the equipment, and cleaning.[32] Generally speaking, the majority of his time spent in the control room during this timeframe was watching ship traffic and raising and lowering the bridge.[33]

13. When he returned from the Navy, Mr. Coffin went back to work at MCR; he first went to work at a place called the Waterville Shops in Waterville, Maine for four to six weeks doing electrical work in the facility such as in offices and buildings; this was a big repair and maintenance facility for train cars/boxcars with asbestos containing brakes– there would be multiple boxcars under maintenance in the shop.[34] His tasks at the Waterville shops involved installing fluorescent lighting in different section houses, he also did work in the shop where the railroad boxcars would be repaired– he ran conduit and installed wiring for a coke machine in the breakroom, and also climbed poles and replaced outside lighting.[35] There were different crews working on the different components of the boxcars, and Mr. Coffin reported that dust would be generated during repairs inside the main part of the boxcars.[36] He recalled the shop being dusty and dirty and assumes this dust contained asbestos.[37] Although he did not have a specific recollection of having observed anyone working on brakes on the boxcars when he was doing his tasks in the shop, he reported that the boxcars would have required brake work and maintenance for wear.[38] He also recalled seeing air hoses being used in the shop on the undercarriage of the box cars, which were the wheels.[39] He spent two days in this shop installing conduits while such work was going on.[40] Regarding the location of the shop, he reported that the shop was just through a

[29] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 155
[30] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 156-157
[31] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 70-72, 77, 335
[32] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 84-87, 91-100, 107, 283
[33] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 299
[34] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 77-78, 276
[35] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 79-80, 262
[36] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 518-519
[37] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 267, 274-275
[38] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 266-267
[39] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 267-269
[40] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 269-270

door into where he worked and that there might have been a short hallway leading directly into the shop; his boss's work area was attached to the building with the shop and he reported, "I was in and out of that building all the time".[41]

14. After being at the Waterville Shops, Mr. Coffin returned to the Carlton Bridge and worked at this bridge until 1987.[42] There would also be times in the wintertime, where he would get laid off periodically and he would go to work at the Rigby yard and the main yard in South Portland, Maine to do snow removal.[43] He reported working swing time for "quite some time" and worked 1 p.m. to 9 p.m. on Mondays and Tuesdays, 9 p.m. to 5 a.m. on Wednesdays, he would get Thursday and Friday off and then on Saturdays and Sundays worked 5 a.m. to 1 p.m.[44] His duties were not any different during these shifts except that he did not do maintenance tasks on the 9 p.m. to 5 a.m. shift, unless it was required.[45] He worked an eight-hour overtime shift every two weeks, but otherwise worked 40-hour weeks.[46]

15. Working as an operator of the bridge, Mr. Coffin spent 90 percent of his time in the control/operating room.[47] He estimated spending an average of once a week in the engine room but did not specify how long he would be in there. While in the control room, he would be watching for boats, and listening to radio traffic of boats coming and going to operate the drawbridge accordingly.[48] On a typical day as a Drawbridge Operator, he monitored traffic (unless there was maintenance scheduled).[49] He recalled that most of the maintenance work entailed greasing components.[50] Besides monitoring the traffic of cars, trains and ships coming down the river. Mr. Coffin described his other regular duties and responsibilities as Drawbridge Operator: greasing components as part of regularly scheduled maintenance, maintaining/replacing navigational lights, stair lights and catwalk lights, maintaining about 80 back-up batteries by periodically adding acid, and general cleaning every couple of weeks.[51]

16. Mr. Coffin testified having assisted his boss in replacing the worn brakes associated with the lift mechanisms on the bridge on two occasions (once in the 1970s and once in the 1980s), as part of an unscheduled maintenance. It took two hours to remove the brakes, replace the pads and put the brakes together.[52] He described how they changed the brakes: they would loosen the shoes off and the braking material was on the inside; replacement braking material was inside the

[41] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 269
[42] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 79-80
[43] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 80-81
[44] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 83
[45] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 84
[46] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 90
[47] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 344-345
[48] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 86-87
[49] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 89
[50] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 90
[51] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 84-87, 90-100, 368-369
[52] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 112

maintenance shop– they would take the shoes to the maintenance shop, put them in a vise, they were held by 10-12 rivets, they would drill those out, pull out the leftover padding, they would cut out the padding material with a hacksaw, cut it to fit, drill it and put rivets and peen them over and put it back together– the replacement braking material was tannish gray and was stored as a roll of 10 feet material.[53] He did not know the brand of friction material they used for this work.[54] He estimated the dimension of each brake shoe to be four inches wide and 16-18 inches long.[55] The breaking system was inside the engine room.[56] When they removed the brake, he reported there was dust from use; the room where the motor was located was described as dusty and greasy.[57] He also recalled that since they had to rivet the lining back on the brake shoes, there would be dust generated during drilling with a hammer.[58]

17. Mr. Coffin also reported having replaced the brake material on the braking system of the barrier gate about four times by himself during his entire tenure.[59] There was one brake per barrier gate and two gates on each end of the bridge; there were two linings per brake.[60] He recalled doing this much the same way– with the same friction material that he cut with a hacksaw as that of the brakes for the motors, except that these were much smaller brakes– three by five inches.[61] It took him an hour and a half to two hours to change a brake on the barrier gates, it took seconds to cut the new friction material and four to five minutes to remove the old friction material; 30-45 minutes to install the new friction material since he would have to re-rivet it by hand onto the shoe.[62]

Maine Department of Transportation (MDOT) September 1987–July 1988

18. Mr. Coffin remained a Drawbridge Operator for MCR until September 1987 when the Maine DOT took over and gave him the Chief Bridge Operator title; he was employed by the DOT for approximately 10-months after which he retired from the railroad.[63] As a Chief Bridge Operator, he also had administrative duties like keeping time sheets and training new people, in addition to his typical duties he would have done as a Drawbridge Operator.[64]

[53] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 114, 366-367
[54] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 114
[55] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 361
[56] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 362
[57] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 368
[58] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 369
[59] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 117, 288
[60] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 292-293
[61] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, 117-119, 291, 371
[62] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 291-292
[63] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 82, 160
[64] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 82-83

The Carlton Bridge

19. The Carlton bridge opened for train traffic– lower deck in October 1927 and officially opened to automobile traffic– upper deck on November 15, 1927.[65] The control/operating room, where Mr. Coffin operated the draw itself to raise and lower the bridge, was a two-story room located on the tower above the street and went all the way across the bridge from side to side.[66] From the ground floor to the top, the control room was about 20 feet high; on the bottom section of the control/operating room was where the span locks were located to lift the bridge.[67] The backup batteries and the maintenance/machine shop was located on the top story where there were tools, vices and grinding wheels; the maintenance shop was the same dimension as the operating room, about 10 by 20 ft with 10 ft ceiling; the windows in the maintenance shop were never opened but they always kept the door open; he reported that there was no ventilation in the maintenance shop.[68]

20. Mr. Coffin reported having done a lot of work in the maintenance/machine shop, including maintenance work on the contact knife blades inside the rail locks, which involved removing them and using the grinding wheel with a wire brush on one end to clean the contacts once every couple of weeks.[69] There were also contacts from an electrical panel in the control room that he would have to remove and smooth out the surfaces with the grinding wheel and reinstall.[70] He reported that he did not think the grinding wheels contained asbestos.[71]

21. Beneath the middle of the span or beneath the roadway is the engine room where the motors were located to lift and lower the bridge.[72] The breaking system was also inside the engine room.[73] Mr. Coffin recalled the engines were electric and were approximately four feet long, three feet high and provided the power to turn the shaft to turn the spool that pulls the cables down, which would lift the bridge up and also lower the bridge back down; the brakes for the engines were separate from the motors.[74] Mr. Coffin believes there was asbestos on the exposed brakes for the electric motors, since it required a very large braking system– the span weighed 270 tons; when the brakes were used, it would slow down the bridge and produce dust from stopping, due to wear and friction.[75] He explained the braking mechanism: there was a drum and two brakes

[65] https://www.mainememory.net/artifact/27897
[66] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 101
[67] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 106
[68] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 106, 280-282
[69] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 107, 283
[70] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 284-285
[71] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 341
[72] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 108-109
[73] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 362
[74] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 110
[75] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 13-14

wrapped around the drum, one on each side; when he would apply the brakes, it would press on the drum and slow the motor down which would then slow the bridge down, producing dust.[76]

Use of Asbestos-Containing Material (ACM) on the Carlton Bridge

22. Mr. Coffin testified that at some point after he left the bridge in 1988, he had heard about asbestos remediation that took place in 2001 at the Carlton Bridge related to the engine room, control room, and the machine shop.[77] He recalled that there was also an outhouse down on the tracks that was built using material similar to the control room.[78]

23. According to bidding documents for asbestos abatement work at the Carlton Bridge in Bath, Maine, in 2001, the storage shed, control room, and machinery house had some asbestos containing materials ("ACM"). Based on Asbestos Disposal & Documentation forms provided from the MDOT remediation in 2001, friable and non-friable wrapped ACM was removed from the Carlton Bridge; however, these forms do not specify the location of each of those materials.[79]

24. OSHA defined friability to mean "that the material can be crumbled with hand pressure and is therefore likely to emit fibers."[80] They further clarify by providing examples to qualify that the "fibrous fluffy sprayed-on materials used for fireproofing, insulation, or sound proofing are considered to be friable, and they readily release airborne fibers if disturbed. Materials such as vinyl-asbestos floor tile or roofing felt are considered non-friable if intact and generally do not emit airborne fibers unless subjected to sanding, sawing and other aggressive operations. Asbestos-cement pipe or sheet can emit airborne fibers if the materials are cut or sawed, or if they are broken." The United States EPA in its document entitled "Asbestos NESHAP Regulated Asbestos-Containing Materials Guidance" specifically categorizes cement siding, transite board shingles, etc. as nonfriable ACMs.[81]

25. Mr. Coffin described the construction of the control room as being made up of individual panels with a one-inch strip of trim covering each of the seams.[82] This had been painted multiple times over the course of occupancy. This type of construction is consistent with the typical use of cement panels, even to this day, where control joints (sometimes referred to as expansion joints) are used to account for potential expansion under various weather conditions and possible

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[76] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 14
[77] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 328-329, 504-505
[78] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 506
[79] Materials Produced in the Matter, WC Exhibit 6, 7 and 9-12 (03223292xAE394)
[80] OSHA 1926 *Substance Technical Information for Asbestos - Non-Mandatory. Toxic and Hazardous Substances, Subpart Z*, Safety and Health Regulations for Construction
[81] Shafer, R., et al. 1990 *Asbestos/NESHAP regulated asbestos-containing-materials guidance*. No. PB-91-218701/XAB. Alliance Technologies Corp., Chapel Hill, NC (United States), 1990.
[82] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 356, 508-509

vibration and thereby avoid physical damage to the rigid panels. In the deposition testimony of Michael Brodhead he describes typical construction he has observed of transite sheet installation where "Typically the sheets of transite would be arranged vertically with a gap -- a -they call it an expansion gap or a vibration proof gap between the edges of each sheet, so that they didn't rub together. And that was less of I think a concern about creating fiber dust; as more as a just creating a – an abrasion issue when these were originally installed. And then over the top of that gap was usually placed a – a cover section."[83]

26. Mr. Coffin related in his deposition that trains would be coming with loads of concrete from a cement plant– the Thomaston Cement Plant in Rockland, Maine and the bridge would shake as the train passed. Although the walls/panels would vibrate, Mr. Coffin did not observe any dust resulting from this movement, nor did he ever have to do any clean-up within the control room or other internal spaces, associated with this movement of the wall panels.[84] Therefore, it appears that the lack of dust being generated from the overall movement of the individual panels, as well as the lack of physical damage occurring to the individual panels and the maintenance of the physical integrity of the various rooms on the bridge, demonstrate that the control joints were effective in achieving their intended design purpose.

27. A health and safety inspection form prepared by the state of Maine on February 16, 1984 found very few items of concern on the Carlton Bridge.[85] Within the "Operator's Room" they noted an electrical panel with exposed electrical connections. This memo did not note any damage or deterioration of the internal wall panels or mention the possibility of potential exposure to asbestos to occupants.

28. Mr. Coffin recalled being present in the 1980s during work involving replacement of a leaking lead pipe inside the walls of the maintenance room, associated with capturing roof water; a crew tore out a section of ceiling and wall to get to the pipe in order to replace it; the work took three to four hours and the material removed for the work was about two feet by four feet.[86] There was dust created during this work and Mr. Coffin reported having been present in the room during this work.[87] He did not recall having seen insulation material inside the wall when the work was getting done.[88] He reported having cleaned up after the work was done and recalled taking a brush and brushing the dust off the tops of the batteries and sweeping the floor; cleaning up took him about 20 minutes.[89]

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[83] Stephen Broadhead's Hearing Transcript for the State of Maine Worker's Compensation dated November 27, 2018, page 17.
[84] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 356, 508-509
[85] State of Maine Inter-Departmental Memorandum. 1984.Inspection of the Carlton Bridge – Bath/Woolwich. February 16, 1984.
[86] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 329-330, 507
[87] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 330-331
[88] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 375
[89] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 509-510

Proximity of Carlton Bridge to Bath Iron Works

29. Mr. Coffin noted that the Bath Iron Works (BIW) was located near the bridge, within a quarter of a mile, according to Mr. Coffin's approximation.[90] Mr. Coffin reported that during the summer months, the windows were all open in the control room and wind would generally come from the south and BIW was south of the Bridge.[91] Mr. Coffin believes that he could have been exposed to the airborne asbestos fibers drifting from BIW when they worked in the hulls of ships.[92] Naval ships often utilized the far more potent forms of amphibole asbestos, primarily amosite, extensively throughout their ships.

U.S. Postal Service Work 1988–2012

30. Mr. Coffin started working for the post office in 1988 as a rural letter carrier in Brunswick, Maine and worked until 2012, when he retired.[93] He reported having used his personal vehicles, which were Subaru's for this job until the last two years when he was provided with a postal vehicle.[94] He recalled having to change all four brakes; he did not report how often he did this.[95] It took him an hour to change the front brakes for a Subaru.[96]

31. The presence of ACM has been confirmed for the Brunswick Post Office location in inspection/assessment documents prepared by URS Corporation and comprised of the following asbestos containing materials in the building: pipe fittings (20% chrysotile asbestos); floor tiles (5-10% chrysotile asbestos); floor tile mastic (3-8% chrysotile asbestos); ceiling tiles (10% amosite asbestos); floor tiles (30-40% amosite asbestos).[97] In addition, according to the Northeast Test Consultants report, three asbestos abatement actions were conducted at the Brunswick post office in 1998, 1999, and 2000 and included removal of amosite asbestos ceiling tiles and chrysotile asbestos mastic material.[98] Per Stephen Broadhead's report dated August 17, 2018, pertaining to ACM in the Brunswick Post office, the building during Mr. Coffin's employment period, contained both friable and non-friable asbestos materials, and non-friable materials that could be impacted into a friable state such as floor tiles by the use of abrasive floor buffing, foot traffic, and constant impact by such actions as rolling mail carts and dollies over the surfaces. In addition, Broadhead reported on the probability of asbestos fiber contamination from vibrating amosite

[90] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 318-319
[91] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 319
[92] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 320
[93] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 160-161
[94] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 162, 447
[95] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 447
[96] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 458
[97] Report by Northeast Test Consultants titled Professional Review for Asbestos Exposure relating to Victor A. Coffin v. State of Maine/Dept of Transportation Dated August 17, 2018
[98] Report by Northeast Test Consultants titled Professional Review for Asbestos Exposure relating to Victor A. Coffin v. State of Maine/Dept of Transportation Dated August 17, 2018

containing ceiling tile systems that subsequently could have been deposited on surfaces and materials located within the mail processing areas of the building, that would then be handled by Mr. Coffin during his rural route delivery duties.[99]

Home Remodeling Work

32. While growing up, Mr. Coffin recalled helping his father move walls around in their house and put in a bathroom sometime in the late 1950s using sheetrock and 2 x 4's.[100] His father would tear the walls down and he would carry them out.[101] He recalled that he did not help with application of joint compound, which he recalled came as a dry product that had to be mixed.[102] Although he did not help, he was near the joint compound being mixed; he recalled the presence of dust when it was poured.[103]

33. The attached garage in their home was torn down and a separate two-car garage built when the plaintiff was around 8-10 years old in the late 1950s.[104] He helped his father by picking up the related construction debris; he recalled that his father removed the asbestos siding–white in color with gray interior, saved it and put it back on the new garage.[105] Other material he recalled during this renovation was wood and insulation; he did not know what type of insulation, except that it was "black crispy."[106] His father had to use a cutter to cut the siding to reuse on the new garage; he recalled having helped during the teardown and rebuilding of the garage and recalled minimal dust generation during this process.[107] It took a couple days to take the shingles off from the old building, stack them up, his father tore down the structure, constructed a two car garage and then put the shingles back.[108]

Work While Building Personal Residence

34. Mr. Coffin reported personally building his first home in 1975/1976 in Topsham Maine– it took six to eight months to build the house.[109] Besides hiring contractors to build the

[99] Report by Northeast Test Consultants titled Professional Review for Asbestos Exposure relating to Victor A. Coffin v. State of Maine/Dept of Transportation Dated August 17, 2018
[100] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 27-28
[101] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 29, 33
[102] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 30
[103] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 31
[104] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 23-24
[105] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 24, 377-378
[106] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 25
[107] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 25-27, 380
[108] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 380
[109] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 175, 177, 216

foundation, install flooring– including Armstrong solarium inlaid vinyl flooring in the kitchen and sheet-rocking–Mr. Coffin did the remaining construction work, installed all the electrical, plumbing and heating system himself.[110] He reported being in the house during installation of sheetrock.[111] The siding he used for the house was Masonite and wood; board and batting on the front with the rest being Masonite siding.[112] He installed using regular asphalt shingles for the roof.[113] Mr. Coffin recalled installing a New Yorker Boiler and Beckett burner.[114] He also reported having replaced a circulator pump four to five years later as well as a rope type gasket that came off around the door to the firebox which he also personally replaced.[115] During the 17 years he lived at this house, he added bedrooms and finished the basement; he would hang the sheetrock and hire contractors to tape joints.[116]

Work as Masonry Tender for Supplemental Income

35. During the winter layoffs at Maine Central, for supplemental income, Mr. Coffin was a masonry tender for a friend of his who owned a business called Dale Wallace & Sons Concrete Masonry, this was on a part-time, sporadic basis for a period of six to eight years in the early to late 1980s.[117] He stated that he worked with refractory cement which he reported contained asbestos.[118] He estimated having worked with refractory cement a total of half a dozen times; it came either in powder form, that would have to be poured and mixed with liquid, or it came in a bucket like drywall mud.[119]

36. The type of masonry work he did involved foundations, fireplaces, chimneys, floors, for mostly residential clients (75 percent) and 25 percent commercial clients such as schools and businesses.[120] Mr. Coffin recalled one job in which they had to remove a large cast iron furnace with insulation "encased in white", which he now believes was asbestos; they used a sledgehammer to break out the insulation which took them one to one and a half hours and then about two hours to carry it out and clean-up.[121]

[110] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 175-178
[111] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 177
[112] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 180
[113] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 180-181
[114] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 179, 228-231
[115] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 217, 226-228, 238
[116] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 181-182
[117] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 163-164, 298-299
[118] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 163, 169-173
[119] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 170
[120] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 164-165
[121] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 168

Home Automotive Work from 1964–2003

37. Around 1964, when Mr. Coffin was 15, he started working on cars, including his own and that of his parents and other family and friends.[122] He reported having owned approximately 24 cars and that he would have done various work on these cars.[123] He has reportedly never owned a new car in his life; all the auto work would have been on used cars.[124] This auto work involved rebuilding transmissions and engines, changing oil, changing brakes and exhaust systems, clutch work and gasket related work.[125] He also recalled having rebuilt a 327 Chevy engine one time on his pickup truck around 1973 or 1974.[126]

Brake Jobs

38. The plaintiff reported having changed brake pads, mostly on the front brakes.[127] He did brake work on both drum style and disc style and recalled Raybestos and Bendix brakes among others.[128] For each drum that he worked on, he would use an air hose to blow off dust, which only took a matter of seconds.[129] He reported that he never roughed up the padding with sandpaper.[130] All of the brake work he did was in driveways and garages.[131]

39. Mr. Coffin recalled the types of cars in which he had worked on brakes: '65 Mustang, '68 Dodge pickup, a bunch of Subaru's, '65 Chevy, '68 pickup truck, Volkswagen Beetle and Rabbit, '68 Chevelle, MGB, MG Midget, Ford Ranger.[132] He did 8-10 brake jobs on his parents' car during the time he was 15 up until he left the house. He recalled another 8-10 total occasions he would have done/helped with brake work on his friends and brother's cars.[133] He reported that he could easily do one brake job a year on his own vehicles up until 2003; also, during the time that he was a mailman and used his own vehicle from 1988 to 2010, he did two or three brake jobs a year.[134]

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[122] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 46, 48, 52
[123] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 50
[124] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 64
[125] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 47, 49
[126] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 48
[127] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 447-449
[128] Deposition of Victor Coffin, Volumes 1 & 2, September 17 and 18, 2018, page 54, 439
[129] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 454
[130] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 455
[131] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 464
[132] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 441-444
[133] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 61-62
[134] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 58, 60

Clutch Replacement Jobs

40. The first time Mr. Coffin did clutch work was on his MGB sports car in which he put in two clutches in 1968/1969 timeframe; over the 35 years he owned this car, he recalled having put in three or four total clutches.[135] He reported having done clutch work less than a total of 10 times during his lifetime.[136] The last time he would have done any clutch work would have been in 1973/1974 on a Toyota Land Cruiser.[137]

Gasket Jobs

41. Mr. Coffin's work on gaskets involved head gaskets and exhaust gaskets on the manifold.[138] He also worked on donut type gaskets between sections of the exhaust system, between a catalytic converter and the muffler.[139] He estimated having done a total of 20-25 gasket related jobs; he only recalled having worked with Feltmate gasket but could not recall other brands.[140] He reported that Feltmate gasket came in a kit and that everything came precut and predrilled and ready to install.[141]

EXPOSURE AND POTENTIAL RISKS WILL VARY SUBSTANTIALLY AMONG INDIVIDUALS BASED ON MULTIPLE CHARACTERISTICS

42. There are many meaningful factors to be assessed that can influence an individual's exposure, dose received, and potential risk to asbestos. These can include but are not limited to:

- Work history of the subject
- Exposure to various chemicals/materials/physical hazards associated with the risk of developing mesothelioma
- Age at exposure
- Magnitude of exposure
- Duration and frequency of exposures
- Typical respiratory rate associated with relevant tasks
- Defining realistic exposure scenarios that are applicable to the subject
- Identifying epidemiological studies that support the disease outcome (mesothelioma) at the concentrations encountered by the subject

[135] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 66
[136] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 63-64
[137] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 65
[138] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 66
[139] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 67
[140] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 68
[141] Deposition of Victor Coffin, Volume 1, September 17, 2018, page 69

- Assessment of possible other exposure scenarios

Chemical Characteristics/Types of Asbestos

43. Asbestos is a generic term used to describe a subset of crystalline mineral silicate fibers that vary in crystal structure and mineral content.[142] These include chrysotile, which is a serpentine mineral as well as five amphibole minerals– amosite, crocidolite, actinolite, tremolite, and anthophyllite.[143] The structure of these silicate minerals can be fibrous or non-fibrous. When they have relatively large ratios of length to width, they are considered to be in the "asbestiform" habit.[144] The type (chemical nature), concentration, and dimensions of asbestos fibers greatly influence the risk of developing asbestos-related disease. There are specific exposure concentrations for various asbestos types that will pose an insignificant risk upon inhalation. Hodgson and Darnton (2000)[145] did an extensive review of mortality reports on asbestos-exposed populations and concluded that the relative potency for causing mesothelioma by the commercial asbestos types, chrysotile, amosite, and crocidolite, is in the ratio of 1:100:500 respectively. Their estimate also assumed that the commercial chrysotile may be contaminated by tremolite. Berman and Crump (1995)[146] also showed that the best estimate for the potency of chrysotile for causing mesothelioma to be hundreds of times less than that of the amphiboles.

44. A detailed review of the exposure-response literature has been published, that looked primarily at chrysotile-exposed cohorts in industrial settings to evaluate a "no-effect" exposure level for development of lung cancer and mesothelioma. The authors found that a cumulative exposure range of 15 – 500 fibers per cubic centimeter per year (f/cc-yr.) is a useful surrogate for a no observed adverse effect level (NOAEL) for the potential risk of developing asbestos related mesothelioma of the lung.[147] With respect to mesothelioma, Berman and Crump reported that the best estimates for the relative potency of chrysotile ranged from zero to about 1/200th that of amphibole asbestos.[148]

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[142] Lippmann M. 2014.Toxicological and epidemiological studies on effects of airborne fibers: Coherence and public health implications. *Critical Reviews in Toxicology*, 44:8,643-695

[143] International Agency for Research on Cancer. 2012. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 100C. Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite)

[144] Lippmann M. 2014. Toxicological and epidemiological studies on effects of airborne fibers: Coherence and public health implications. *Critical Reviews in Toxicology*, 44:8,643-695

[145] Hodgson JT, Darnton A. 2000. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Annals of Occupational Hygiene*, 44:565-601.

[146] Berman DW, Crump KS, Chatfield EJ, Davis JM, Jones AD. 1995. The sizes, shapes, and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation. *Risk Analysis*, 15(2):181-95.

[147] Pierce JS, McKinley MA, Paustenbach DJ, Finley BL. 2008. An evaluation of reported no-effect Chrysotile asbestos exposures for lung cancer and mesothelioma. *Critical Reviews in Toxicology*, 38:191-214.

[148] Berman DW, Crump KS. 2008. Update of potency factors for asbestos-related lung cancer and mesothelioma, *Critical Reviews in Toxicology*, 38:sup1, 1-47.

45. The fiber type historically used in asbestos cement boards, brake pads, and engine gaskets was chrysotile.[149,150] Yarborough's review of exposure to high concentrations of chrysotile fibers not contaminated with amphiboles did not support a conclusion of causation.[151] As described above, chrysotile is generally considered to present a negligible risk for development of mesothelioma, as opposed to the other commercially available forms of asbestos, which are amphiboles.

Review of the Relevant Scientific Literature with Respect to Incidental Exposure to Asbestos Cement Board Dust, Performance of Mechanic Work on Specific Mechanical Components, and Development of Cancer or Mesothelioma Due to Exposure to Asbestos

46. My review of the available published literature reveals no case studies or exposure assessments specific to incidental/bystander exposures to asbestos containing cement board dust or mechanic service work, associated with heavy equipment and asbestos found in their associated brake pads. Governmental agencies and researchers have carried out exhaustive studies since the 1960s to identify those products, activities, and environments that contained asbestos, created an airborne exposure to asbestos, and presented a risk to those populations that would encounter it. This included evaluating direct workplace exposures and those that are termed "bystander" exposures, whether they occurred in an occupational setting, in the community, or in a "take home" residential setting. The focus of the scientific literature reflects that workers and residents in asbestos cement containing structures are not at risk for consequential exposure as a result of routine activities.

47. My review of the literature also found that the preponderance of epidemiological studies clearly showed no increased rates of mesothelioma, among full-time mechanics servicing automobiles and trucks, that routinely performed brake replacements and/or gasket replacements, as part of the engine rebuilding process.[152,153,154] Mr. Coffin's incidental exposure and very limited active work during his time as a Bridge Operator, at the Carlton Bridge, would be even less of a risk to provide any potential exposures that would be of physiological significance, since they

[149] Jacko MG, DuCharme RT. 1973. Brake emissions: Emission measurements from brake and clutch linings from selected mobile sources. Conference: National Automobile Engineering Meeting, February 1973.

[150] Blake CL, Dotson GS, Harbison RD. 2006. Assessment of airborne asbestos exposure during the servicing and handling of automobile asbestos-containing gaskets. *Regulatory Toxicology and Pharmacology*, 45(2):214-22.

[151] Yarborough CM. 2007. The Risk of Mesothelioma from Exposure to Chrysotile Asbestos. *Current Opinion in Pulmonary Medicine*, 13(4):334-8.

[152] Teschke K, Morgan MS, Checkoway H, Franklin G, Spinelli JJ, van Belle G, Weiss NS. 1997. Mesothelioma surveillance to locate sources of exposure to asbestos. *Canadian Journal of Public Health*. 88 (1)163 – 168.

[153] Garabrant, DH, Alexander DD, Miller PE, Fryzek JP, Boffetta P, Teta MJ, Hessel PA, Craven VA, Kelsh MA, Goodman M. 2016. Mesothelioma among motor vehicle mechanics: An updated review and meta-analysis. *Ann. Occup. Hyg* 60(1):8-26.

[154] Laden F, Stampfer MJ, Walker AM. 2004. Lung cancer and mesothelioma among male automobile mechanics: A review. *Reviews on Environmental Health*. 19(1)39-61.

would be so much lower and infrequent when compared to those previously cited assessments of full-time mechanics, who were directly and routinely servicing large numbers of vehicles.

Exposure Factors Relevant to Mr. Victor Coffin and Reported Exposures Associated with His Time Working as a Bridge Operator

48. Several published articles have been reviewed that report on personal and area air samples collected during periods of time when heavy equipment and heavy truck brake pads were removed and replaced. Although not specifically related to bridge braking systems, the various heavy equipment exposure characterizations are analogous to possible exposures that would be encountered during typical brake repair activities associated with bridge equipment.

49. Of the published reports reviewing the presence of fibers in the workplace, I will utilize two in developing the potential exposure scenarios relevant to Mr. Coffin's work experience on the Carlton Bridge. One summarized the relevant literature evaluating possible exposure to heavy equipment mechanics from brake dust, during change out and replacement, while another assessed the potential exposures during servicing of automobile asbestos containing brakes, which would be a "worst-case" analogy for servicing the barrier brakes he described.[155,156] These studies are consistent with others found in the published literature[157,158,159] that showed that exposures to workers and mechanics that regularly undertook these types of repairs on heavy equipment and heavy trucks, were far below any levels that would be considered hazardous, even when compared against the most stringent exposure control guidelines and standards.

50. Mr. Coffin also related that he was present in the Machine Room when other workers removed a section of ceiling/wall that was approximately two feet by four feet to repair a leaking pipe. Mr. Coffin was not the primary individual performing the work on the removal and repair of AC board wall panels, but rather he was an observer/bystander of the work activities by others[160] and only indirectly participated and "helped with the clean-up".[161,162] For example, in a study where AC board was removed from a wall structure, the bystander's exposure (the subject was within 1.5 meters of the work activity) during the active cutting and removal of the wall board,

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155 Paustenbach DJ, Ritchter RO, Finley BL, Sheehan PJ. 2003. An evaluation of the historical exposures of mechanics to asbestos in brake dust. *Applied Occupational and Environmental Hygiene* 18:786-804.

156 Madl AK, Gafeney SH, Balzer JL, Paustenbach DJ. 2009. Airborne Asbestos Concentrations Associated with Heavy Equipment Brake Removal. *Annual Occupational Hygiene* 53 (8) 839-857.

157 Spencer JW, Plisko MJ, Balzer L.1999. Asbestos Fiber Release from the Brake Pads of Overhead Industrial Cranes. *Applied Occupational and Environmental Hygiene* 14 (6) 397 – 402.

158 Blake CL, Van Orden DR, Banasik M, Harbison RD. 2003. Airborne asbestos concentration from brake changing does not exceed permissible exposure limit. *Regulatory Toxicology and Pharmacology,*38(1):58-70.

159 Spencer JW, Plisko MJ, Balzer L.1999. Asbestos Fiber Release from the Brake Pads of Overhead Industrial Cranes. *Applied Occupational and Environmental Hygiene* 14 (6).

160 Deposition of Victor Coffin, Volume 2, September 18, 2018, page 329-330, 507

161. Deposition of Victor Coffin, Volume 2, September 18, 2018, page 330-331

162 Deposition of Victor Coffin, Volume 2, September 18, 2018, page 509-510

ranged between 0.01 and 0.02 f/cc, during the active work period. The personal sample obtained on an individual during the clean-up activity was 0.9 f/cc.[163] This simulation would likely be higher than Mr. Coffin's general clean-up exposure since this simulation had the subject collecting and bagging a significant amount of freshly cut AC board debris, as opposed to the very limited exposure Mr. Coffin described in his deposition.

51. Therefore, based on information provided by Mr. Coffin in his deposition, the scenarios that would describe how the Plaintiff could be potentially exposed to asbestos, originating from his activities associated with his time of employment for the Maine Central Railroad during the time period of the second half of 1967 and between 1971 and 1987, would be:

a.) Being present in a physical structure (Control Room, Engine Room and Maintenance Room) constructed of AC boards.
b.) A "bystander exposure" while being present and in a room where AC boards were removed, and then performing clean-up of the room for approximately 20 minutes after completion of the removal task by others.
c.) Assisting with the replacement of brake pads on the bridge lift mechanism, twice.
d.) Replacement of each of the four "barrier" brakes, four times.

RESULTS OF AN EXPOSURE MODEL SPECIFIC FOR MR. COFFIN'S STATED HISTORY OF WORK FOR MCR AND DEMONSTRATED NO AIRBORNE HAZARD EXISTED

Working in a Structure Constructed of AC Boards
Bystander Exposure for Engine Rebuilding and Brake Replacement

52. The exposure reconstruction of relevant work performed by the Plaintiff is based on the testimony of Mr. Coffin. It relates to the time period of the second half of 1967 and between 1971 and 1988, and can be summarized as follows:

Time period- The Plaintiff was a Bridge Operator for approximately 17.5 years. During that time period he would spend the majority of his time in the various structures associated with the Carlton Bridge, although he did routinely perform service repairs outside of those environments.

Frequency- Although Mr. Coffin was not specific in the number of months he worked on the Carlton Bridge each year, his deposition testimony states that he was there nearly full-time, except for periods during the winter when he would be laid off, or transferred back to the Rigby or the

[163] ASEA Reports. 2016. Measurement of asbestos fibre release during removal works in a variety of DIY scenarios. Prepared by Monash University. Asbestos Safety and Eradication Agency, Australian Government.

Main Yard, for an unspecified period of time. To be conservative, we will assume that Mr. Coffin worked on the Carlton Bridge for a maximum of 46 weeks per year (assuming 10 days vacation, 10 days holiday and 10 days of winter furlough, or other temporary assignment) to develop a "high" potential exposure scenario.

Duration- Although there is no specific information provided by Mr. Coffin as to how long his required maintenance on the bridge would require him to be out of the various bridge enclosures, we will assume that he spent 100 percent of his time within the structures, and this will be utilized in these calculations because it will contribute to providing a highly conservative exposure estimate.

Magnitude of Exposure- No documents were identified that discussed exposure to asbestos fibers from AC board constructed buildings. The structure under consideration was placed into service in 1927 and had been continuously used until it was decommissioned in approximately 2000. The building had been in continuous use for more than 40 years before Mr. Coffin first worked there. There was no record of it requiring repair or replacement during that time period. The building, as described by Mr. Coffin, was constructed in accordance with reasonable and prevailing building practices of the time, including a double wall, frame construction. Mr. Coffin did not cite any damage to the walls of the structure, and further cited that the walls had been painted several times and were reported to be smooth and in good condition.[164] The painted surfaces would serve to further encapsulate the asbestos contained in the AC boards. Mr. Coffin discussed vibration of the walls related to railway and automobile traffic, and in hindsight wondered if the vibration could have potentially caused the AC boards to rub against each other and release fibers. There is no evidence presented that indicated that this action ever occurred, or that the panels had any friable materials present. The seams between the panels were not visible to Mr. Coffin, since he testified that they were covered with a piece of trim.[165] Mr. Coffin's recollection of cleaning dust in the enclosures cannot be used to demonstrate that asbestos fibers were released, or present, since "dust" is not a specific indicator for asbestos fibers and is very prevalent along rail tracks and roadways; generic roadway dust can be suspended in air by the motion of vehicular or train traffic and may have nothing to do with asbestos releases from a structure. However, as done previously, I have assumed a "worst case" scenario to test the hypothesis that Mr. Coffin may have been exposed to asbestos fibers during his work on the Carlton Bridge. The "high" estimate was considered to be the average of urban background (0.0005 f/cc)[166] and the "low" estimate was the median for rural background (0.00002 f/cc).

[164] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 508
[165] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 508
[166] Abelmann, et al., 2015 Historical ambient airborne asbestos concentrations in the United States - an analysis of published and unpublished literature (1960s-2000s). Inhal Toxicol. 2015;27(14):754-66.

Direct Exposure for Reported Brake Replacement

53. Madl et al.,[167] evaluated the potential exposure to individuals that were doing regular brake jobs on automobiles and heavy equipment. These can be used as analogous exposure estimates for Mr. Coffin's work on the brakes on Carlton Bridge. Therefore, the magnitude of direct exposure for an individual, like Mr. Coffin during the removal and replacement of brake pads on the Barrier Gates will be estimated by those studies associated with automobile brakes, and the brake mechanism for the draw bridge, by use of brake replacement on heavy equipment.

Direct Exposure Scenario (lift mechanism brake replacement)- high: This would involve being present for two lift mechanism brake replacements for a duration of two hours each at an exposure of 0.09 f/cc.

Direct Exposure Scenario (lift mechanism brake gasket replacement)- low: This would involve being present for two brake replacements for a duration of two hours each at an exposure of 0.044 f/cc.

Direct Exposure Scenario (barrier gate brake replacement)- low: This would involve being present for four brake replacements during his employment for a duration of one and a half to two hours each at both ends of the bridge at a worker personal concentration of 0.03 f/cc.

Direct Exposure Scenario (barrier gate brake replacement)- high: This would involve being present for four brake replacements during his employment, for a duration of one and a half to two hours each at both ends of the bridge at a worker personal concentration of 0.09 f/cc.
<u>Table One</u>- Estimates for lifetime direct cumulative dose associated with Mr. Coffin's work as a Bridge Operator for MCR on the Carlton Bridge compared to lifetime dose associated with the 1986 OSHA PEL of 0.2 f/cc and the current OSHA 8-hour PEL of 0.1 f/cc exposure to asbestos.

[167] Madl AK, Gafeney SH, Balzer JL, Paustenbach DJ. 2009. Airborne Asbestos Concentrations Associated with Heavy Equipment Brake Removal. *Annals of Occupational Hygiene,* 53 (8) 839-857.

Task	Time Period (Weeks)	Frequency of Event (Days/Week)	Duration of Event (Hours/day)	Exposure Factor (f/cc)	Cumulative Exposure (f/cc-hr.)	Cumulative Exposure (f/cc-yr.)
Brake Replacement						
Lift (H)	2	1	2	0.09	0.36	0.00018
Lift (L)	2	1	2	0.044	0.176	0.00009
B. Gates (H)	4	1	4	0.090	1.44	0.00072
B. Gates (L)	4	1	4	0.030	0.48	0.00024
Maintenance Room Repair Work						
Bystander (H)	1	1	4	0.02	0.08	0.00004
Bystander (L)	1	1	4	0.01	0.04	0.00002
Clean-up	1	1	0.33	0.9	0.297	0.00015
Bridge Operator						
Indoor (H)	840	5	8	0.0005	168	0.0084
Indoor (L)	840	5	8	0.00002	0.672	0.00034
Regulations						
1986- 8-hr. PEL (OSHA)	840	5	8	0.2	67,200	33.6
1994- Current 8-hr. PEL (OSHA)	840	5	8	0.1	33,600	16.8

f/cc = fiber per cubic centimeter
hr. = hour
yr. = year
(H) = High Estimate
(L) = Low Estimate
PEL = Permissible Exposure Limit
OSHA = Occupational Safety and Health Administration

Calculating MCR Work Associated Lifetime Exposure (cumulative):

Based on realistic, but highly conservative assumptions regarding the relative time period, frequency, duration, and exposure concentrations, the calculations detailed in Table One result in a cumulative exposure with a range from 0.00084 f/cc-yrs. to 0.00958f/cc-yrs. for the Plaintiff's possible exposure to asbestos containing materials during the 17.5 year period in which Mr. Coffin reportedly worked as a Bridge Operator on the Carlton Bridge.

A comparison of the potential cumulative exposure of the Plaintiff, with occupational exposure limits for airborne asbestos compared to his potential exposure risks associated with asbestos resulting from exposure to asbestos fibers associated with his work as a Bridge Operator, is negligible. As a point of comparison, Mr. Coffin's actual cumulative exposures would have been between 3.500 to 40,000 times lower than the most stringent occupational exposure regulations

and guidelines that were in effect at the time of his employment, as well as 1,700 to 20,000 times below the most stringent regulations that are currently in effect.

Comparison to Regulatory and Professional Standards in Force During Mr. Coffin's Relevant Work Period with MCR

54. The American Conference of Governmental Industrial Hygienists (ACGIH), was established in 1938 (originally known as the National Conference of Governmental Industrial Hygienists), by a group of industrial hygienists working at various state, city, and federal agencies, including the United States Public Health Service. A major charge that they undertook was to review the scientific literature and suggest exposure limits that would be protective of worker health. These exposure limits were widely employed and periodically reviewed as new information became available. They provided guidance for asbestos exposure limits that were widely used throughout industry until OSHA was formed in 1971. These exposure limits, that they termed "Threshold Limit Values" (TLVs), were intended to represent concentrations under which "nearly all workers can be employed for their entire working lifetime without adverse effect."[168]

55. In 1968 the ACGIH published a Notice of Intended Change for its asbestos TLV with a proposal to reduce the standard to 2 million of particles per cubic foot (mppcf) or 12 f/cc as an 8-hour Time Weighted Average (TWA).[169]

56. In 1969, the Walsh Healey Public Contracts Act adopted the 1968 ACGIH TLVs including the Notice of Intended Change for Asbestos of 12 f/cc.

57. In 1970, the ACGIH published a Notice of Intended Change to its asbestos TLV which proposed to reduce the 8-hour TWA to 5 f/cc and to include a "Ceiling Limit" of up to 10 f/cc which could occur for no more than 15 minutes each hour for up to five times in an 8-hour workday.[170]

58. The Occupational Safety and Health Administration (OSHA), was formed in May of 1971 by an act of Congress and set about to develop regulations for limiting workplace exposure to specific chemicals, including asbestos, and termed them Permissible Exposure Limits (PELs). The table below provides a summary of OSHA's regulations for airborne asbestos.

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[168] Proceedings of the 8th Annual Meeting of the ACGIH: TLV Committee Report, Chicago, IL., April 7-13, 1946.
[169] Transactions of the 30th Annual Meeting of the ACGIH, St. Louis, MO. May 12-14, 1968.
[170] Transactions of the 32nd Annual Meeting of the ACGIH, Detroit, MI. May 10-12, 1970.

Table Two History of OSHA Asbestos Standards

Date	Standard	Notes
5/29/1971	12 f/cc TWA	8 hr. TWA
12/7/1971	5 f/cc TWA; 10 f/cc Ceiling	Emergency Temporary Standard (ETS); 8 hr. TWA; 15 min ceiling
6/7/1972	5 f/cc TWA; 10 f/cc Ceiling	8 hr. TWA; 15 min ceiling
7/1/1976	2 f/cc TWA	8 hr. TWA
6/20/1986	0.2 f/cc TWA	8 hr. TWA, Action Level = 0.1 f/cc
9/14/1988	1 f/cc - Ceiling	30 min Excursion Limit
8/19/1994	0.1 f/cc TWA	8 hr. TWA

hr. hour
min minute
OSHA Occupational Safety and Health Administration
f/cc fibers per cubic centimeter
TWA time weighted average
TLV Threshold Limit Value

59. The airborne concentrations calculated and detailed in Table One, used realistic and appropriate exposure scenarios that were developed from Mr. Coffin's deposition testimony and the relevant scientific literature show that the resulting concentrations would fall well below any of the pre-existing 8-hour time weighted averages that were in place at any point during his employment by the MCR. This also includes the current OSHA Permissible Exposure Limit of 0.1 fibers/cc as well as the current OSHA permissible 30-minute Excursion Limit of 1.0 fibers/cc. Further, a sensitivity analysis on this approach demonstrates that the maximum emissions Mr. Coffin would have experienced, as described in his deposition testimony, would still result in an airborne concentration of asbestos that would be well below the OSHA standards that were in place during his employment with the MCR as well as the stringent OSHA guidelines that are currently in place.

Alternative Theories of Causation

60. Mr. Coffin reported significant activities associated with work he performed as a child and as a young man, both with his father and by himself, while doing different types of construction work around the personal residences that utilized the demolition, cutting and reuse of

AC board and shingles, as well as other asbestos containing products. Work in the construction industry has been shown to be associated with an increased risk of developing mesothelioma.[171]

61. Mr. Coffin reportedly did extensive work on military aircraft, both helicopters and planes, during his time in the Navy. It is well established that military aircraft contained asbestos in numerous applications as insulation, soundproofing, and fire protection. Furthermore, military aircraft of the vintage coinciding when Mr. Coffin was in the military had asbestos insulation on its wiring,[172] which Mr. Coffin dealt with on a routine basis while in the service.

62. Mr. Coffin also described how he worked with refractory cement in a commercial setting when he was furloughed during winter months while working for MCR over a period of six to eight years. Refractory cement contains high percentages of asbestos fibers that were primarily chrysotile. Furthermore, Mr. Coffin discussed how he removed a large boiler from a multifamily residence by breaking apart the cement that encased it with a sledge hammer. Mr. Coffin believed that the cement was an asbestos insulating cement. This very physical work would have taken place in extremely close quarters with little ventilation which would have generated significant amounts of respirable dust that likely contained significant amounts of asbestos fibers.

63. Mr. Coffin worked for the U.S. Postal Service out of the Brunswick Facility. Reports provided from that facility show that extensive asbestos containing materials were present in the facility, in a damaged and friable state, prior to the extensive remediation actions that were performed there.[173] The materials were documented to contain both chrysotile and amphibole asbestos.

64. Mr. Coffin also described how he did extensive work on his vehicles throughout his adult life– changing brakes and replacing clutches and engine gaskets.

65. Mr. Coffin noted that the Bath Iron Works (BIW) was located near the bridge, within a quarter of a mile, according to Mr. Coffin's approximation.[174] Mr. Coffin reported that during the summer months, the windows were all open in the control room and wind would generally come from the south and BIW was south of the Bridge.[175] Mr. Coffin believes that he could have been exposed to the airborne asbestos fibers drifting from BIW when they worked in

[171] McDonald AD, McDonald JC. 1980. Malignant Mesothelioma in North America. *Cancer*.46:1650-1656

[172] Advisory Circular. 2013. Aircraft Wiring and Bonding.AC 21-99(1). Annex D to Section 2, Chapter 1, MIL-DTL-25038 wire, Electrical, High Temperature, Fire Resistant and Flight Critical, General Specification. Civil Aviation Safety Authority, Australian Government .

[173] URS Corporation's Asbestos & Lead-Based Paint Compliance Audit Report from March 2005 for Brunswick Post Office.

[174] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 318-319

[175] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 319

the hulls of ships.[176] Naval ships often utilized the far more potent forms of amphibole asbestos, primarily amosite, extensively throughout their ships.

Relevant Dose Response Considerations

66. For virtually every substance, the dose to which an individual is exposed relates to the likelihood that a physiological effect will be observed. Current literature supports the presence of a "threshold dose" for chrysotile, below which there would be no causal risk for developing mesothelioma. The exposures Mr. Coffin may have received from his very limited direct activities associated with being a Bridge Operator on the Carlton Bridge would have been far below the threshold exposure. This is due to the very low cumulative exposure that he could have received as well as the type of asbestos that was present.[177].

67. The Pierce et al., 2008 review[178] summarizes the cumulative exposure-response data reported for predominantly chrysotile-exposed cohorts in the available literature and found that for predominantly chrysotile exposures, the cumulative "no-effects" exposure levels for mesothelioma fell between 15–500 f/cc-yr. Therefore, the No Observed Adverse Effect Level found in the literature was between approximately 1,500 to more than 50,000 times greater than the maximum cumulative exposure value calculated for the Plaintiff, Mr. Coffin, over the maximum period of time that he would have been employed as a Bridge Operator by the MCR.

CONCLUSIONS

68. In addition to the opinions expressed above, my principal conclusions, to a reasonable degree of scientific certainty, include the following:

a) The Plaintiff's incidental exposure to airborne asbestos fibers originating from his work as a Bridge Operator, while employed by MCR was very low and consistent with the lower estimates of normal employment in an industrial setting. None of Mr. Coffin's potential exposures would have exceeded, or approached, the most stringent regulations that were in place at the time of his employment or even those that are in force at this present time.

b) Analysis of relevant data compiled from analogous studies of mechanics and other occupations, application of standard industrial hygiene methodologies, in

[176] Deposition of Victor Coffin, Volume 2, September 18, 2018, page 320
[177] Roggli VL. 2007. Environmental asbestos contamination: What are the risks? *Chest,* 131:336-338
[178] Pierce JS, McKinley MA, Paustenbach DJ, Finley BL. 2008. An evaluation of reported no-effect Chrysotile asbestos exposures for lung cancer and mesothelioma. *Critical Reviews in Toxicology,* 38:191-214.

addition to comparison with results found in published literature, do not support the contention that there is a health hazard associated with the low level asbestos cumulative exposure encountered by the Plaintiff via the incidental exposures, that would have occurred from his work as a Bridge Operator on the Carlton Bridge. This is true even when using extremely conservative assumptions that would tend to bias the calculated results toward a higher potential exposure.

c) Any exposure to asbestos from Mr. Coffin's reported time around the activities associated with brake replacement while working at the Carlton Bridge, would have been far less (by several orders of magnitude) than the potential dose received by full-time mechanics. This is significant since the vast majority of epidemiological studies have shown no statistically meaningful association with automobile, heavy equipment, or heavy truck mechanics and a risk of developing mesothelioma. These epidemiological study findings of no association are very robust due to their consistency between various study designs, their consistency over time (multiple decades), and their consistency between various nationalities and continents.

d) There is no evidence or testimony presented that show/describe that there was physical damage or abrasion to any of the boards/panels that were used to construct the enclosures on the Carlton Bridge. AC board was a common building material that was used in thousands of structures around the globe for decades and due to the immobilization and encapsulation of the asbestos fibers in the body of the board was not, and is not, considered to pose a risk except when it is cut or demolished. The limited cutting that took place on the panels did not present a meaningful risk to Mr. Coffin.

e) The specific type of asbestos that was used in the AC Boards, that were produced in the 1920 to 1930-time frame, was specified to be chrysotile. This form of asbestos has been shown to have a negligible potential to cause mesothelioma as opposed to the amphibole types.

f) There was no information presented in the documentation review that indicated that Mr. Coffin would have been exposed to amphiboles as part of his work at the Carlton Bridge.

In addition to the materials specifically cited above, I reviewed the materials listed in Appendix B for my evaluation and in formulating my opinion.

I reserve the right to supplement my report if additional data or information becomes available in the future.

I declare under penalty of perjury that the foregoing is true and correct.
Executed on January 6, 2020, in Newton, Massachusetts.

By:

John F. McCarthy, ScD., C.I.H.

Attachments:

Appendix A JFM *Curriculum Vitae*

Appendix B Documents Relied Upon for Expert Opinion

APPENDIX A

JOHN F. MCCARTHY, SC.D., C.I.H. – CURRICULUM VITAE

JOHN F. MCCARTHY, SC.D., C.I.H.

PRESIDENT

BACKGROUND SUMMARY

1988 –	President, Environmental Health & Engineering, Inc.
1992 –	Lecturer, Dept. of Environmental Health, Harvard School of Public Health
1980 – 1987	Research Scientist/Director, Aerosol Characterization Laboratory, Massachusetts Institute of Technology
1978 – 1980	Research Scientist, Massachusetts Institute of Technology

EDUCATION

Sc.D. Environmental Science and Physiology, Harvard University, 1987
M.S. Environmental Health Sciences, Harvard University, 1978
B.S. Biology, Boston College, 1973

PROFESSIONAL REGISTRATION

American Board of Industrial Hygiene Certified: Comprehensive Practice

PROFESSIONAL AFFILIATIONS

American Industrial Hygiene Association
International Society of Indoor Air Quality and Climate
American Society for Testing and Materials
American Conference of Governmental Industrial Hygienists
American Society of Healthcare Engineers

COMMITTEE MEMBERSHIP

American Society of Heating, Refrigeration, and Air-Conditioning Engineers, Inc.
IAQ 2004 Conference Chairman

American Society of Heating, Refrigeration, and Air-Conditioning Engineers, Inc.
Guideline Project Committee 10 P, Criteria for Achieving Acceptable Indoor Environments

National Academy of Sciences
Standing Committee on Medical and Epidemiological Aspects of Air Pollution on U.S. Government Employees and their Families

Harvard T.H. Chan School of Public Health
Board of Directors, Center for Global Health and the Environment

EXPERIENCE

As President of Environmental Health & Engineering, Inc. (EH&E), Dr. McCarthy has led investigations for a wide variety of exposures to toxic pollutants and infectious agents and their associated health effects. His work has focused on the analysis of pollutants originating from both outdoor and indoor sources, as well as pollutant transport through various media. He specializes in problem identification and assembly and management of interdisciplinary teams to address the various areas of concern that arise with different clients. Dr. McCarthy provides technical and administrative design direction to the study team, including the development and application of novel analysis techniques and the implementation of field monitoring studies. Relevant air quality experience includes the following:

Ambient Environments

Directed air quality impact assessment of Central Artery Vent Building No. 3 on surrounding community. Performed air quality studies including risk assessment for dispersion modeling of NO_2, CO, and PM_{10} impacts. The work supported an air rights development of property owners and required close interaction with CA/T and Massachusetts Department of Environmental Protection (MADEP).

Provided air quality modeling, risk assessment and atmospheric monitoring of CO, NO_2 and PM_{10} for an apartment/hotel complex to assess potential impact of relocating an access ramp and rerouting vehicular traffic due to CA/T construction.

Provided risk assessment and environmental impact analysis of proposed roadway construction around the Massachusetts General Hospital and Spaulding Rehabilitation Hospital. The work involved negotiating appropriate air quality action levels with MADEP and development of mitigation strategies. As a follow-on project to this, Dr. McCarthy developed and implemented an atmospheric sampling program to verify compliance of the construction program with control measures.

On behalf of the CA/T, principal-in-charge of an air quality impact assessment for the City of Boston's East Boston community during construction of the Third Harbor Tunnel and proposed construction of a hazardous waste incinerator. This involved dispersion modeling and analysis, risk assessment and risk communication.

Principal-in-charge of development of a comprehensive air monitoring program and community impact evaluation for development of a 78-acre contaminated property. In addition to developing a complex monitoring network, this work involved developing appropriate air quality action levels and mitigation measures for permit hearings. This has also involved community liaison and negotiations with state and local authorities.

Directed detailed analysis of the University of Vermont's medical incinerator for an air permit. This work involved assessing possible emissions, providing dispersion analysis, and risk characterization.

For the City of Boston, performed a detailed line source analysis of rerouting traffic due to CA/T activities.

For the City of Cambridge, performed a detailed analysis including atmospheric dispersion analysis and risk assessment for bridge construction and resultant traffic for Charles River span (Scheme Z).

Conducted a detailed analysis of impact of exhaust stack emissions for the University of Cincinnati. This work involved developing a near-field dispersion model, site monitoring to validate performance and making mitigation recommendations.

Principal-in-charge of a detailed analysis and monitoring study (CO, NO_2, $PM_{2.5}$, elemental carbon, speciated VOCs) to characterize exposure of toll collectors to vehicular traffic.
Led a detailed reconstruction of exposure to combustion byproducts of a potentially impacted population using atmospheric modeling after a truck fire which involved hazardous materials. This involved profiling truck contents, estimating emissions rate and dispersion of combustion byproducts and determining estimates of exposure at the receptor locations.

Led the analysis of the environmental impacts from roadway emissions during a major five highway study in Las Vegas, Nevada. This work involved detailed characterization of air toxics, air quality modeling, and identification of highest impact areas. Continuing work involved development and implementation of remedial measures as part of a settlement program.

Product Evaluation

Served as Principal Investigator of a two-year, multi-phased investigation of "Chinese Drywall" conducted on behalf of the U.S. Consumer Product Safety Commission. The investigation included: identification of markers of problem drywall, in-home evaluations of environmental quality and corrosion, health impact analyses, chamber-testing of drywall samples to identify emissions and corrosion rates, evaluations of drywall from domestic (U.S.) suppliers, and an assessment of temporal variability of conditions in homes.

Directed a major product evaluation for a national retailer, manufacturer and importer of clothing to determine formaldehyde emission rates from various clothing materials. The assignment involved determining emissions utilizing controlled environmental chambers under both static and dynamic conditions, performing a sensitivity analysis over a wide range of temperatures and humidities, and recommending limits of acceptability for materials.

For the Commonwealth of Massachusetts, performed a detailed analysis on a number of waterproofing products that had been used in a major state facility that were not performing properly. Through comparison with ambient samples and headspace analysis of various products to identify specific chemical agents, the problem material was isolated and identified. Additional dynamic analyses of building system mockups in environmental chambers permitted precise determination of product cure time to be made.

Developed and oversaw a series of quantitative tests to verify the performance of nano-tube filters designed for use in the pharmaceutical and semi-conductor industries. This program involved evaluating capture efficiency for a range of sub-micron particles as well as the potential for the "shedding" of materials into the airstream during equipment start-up or pulsations in the system.

Principal-in-charge of a study to evaluate the potential release of airborne fibers during maintenance activities of a variety of household appliances. This work involved collecting source, personal and area samples during well-characterized maintenance activities in a large exposure chamber under a range of ventilation conditions. The samples were analyzed using various analytical techniques to permit direct comparison with other historical studies. Project included developing time-activity exposure pattern profiles for maintenance workers.

For a major equipment manufacturer, performed a detailed evaluation of removal of various air pollutants by a new, innovative pollutant removal technology. This involved development of a fully instrumented, 1700 square foot test house/chamber in which several pollutants, including gases, particles, and biological agents, could be precisely released over time and their removal rate accurately monitored. In this multi-phase interventional study a program was designed to determine system efficacy and a corresponding performance model was developed.

For a large urban teaching hospital, developed an evidence based design guide for new orthopedic operating rooms based on control of airborne infectious agents. This involved evaluating the international scientific literature regarding infections from airborne particles, critically reviewing current design guides and their underlying rationale, developing a reproducible test procedure that can be used to characterize the control of potentially infectious particles, and using the testing protocol to compare the effectiveness of the various designs located at multiple institutions.

Developed a controlled chamber test to determine the emission rate of silica and respirable dust from various concrete cutting tools under varying environmental conditions. This test also provided information on particle size distributions associated with various operations.

For a major university, led a multidisciplinary team in developing a detailed characterization study regarding potential exposure to polychlorinated biphenyls (PCBs) used in building materials. This involved a review of plans, specifications and architectural drawings to identify likely areas of use, developing a comprehensive sampling program to verify emission rates and incorporating this into an

exposure model that included various building dynamics parameters such as solar loading, seasonal effects, ventilation patterns, and building pressurization.

Managed a comprehensive testing and analysis program designed to evaluate emissions of radiation from various building materials, including granite and marble for a large national distributor. The work involved developing testing protocols, measuring alpha and gamma emissions from the products under both controlled and real world conditions, and developing exposure estimates for various usage scenarios.

Developed a series of validation tests for fume hood orientations for a large medical research facility. This work involved building a mock-up of the research pod in a controlled test chamber and measuring emissions of a variety of possible contaminants from various operations and procedures that were performed in the containment. These systems were evaluated for various configurations, air exchange rates, and diffuser velocities.

Managed the assessment of emissions of sucrose residue from building materials in an historic structure that was being rehabilitated to a biotechnology research center. This work involved establishing test protocols to evaluate impact environmental conditions such as heat, pH, and moisture. Independent test cells were established at the facility and tests performed in situ.

Managed a program to determine the source of significant noxious odors in a twenty-eight story office tower. By establishing a series of isolated test cells within the building, the odor was isolated to butyric acid residue found in acoustical ceiling tiles located in various areas throughout the building. Additional chamber testing determined the environmental conditions under which butyric acid residue would be released. This data was then used to set allowable limits for acceptable performance during the manufacturing process.

Directed a study for a major hotel chain to evaluate emission of various organic compounds from water that was being processed through a new filtration/aeration system. This work involved developing and implementing a multifactorial study design to evaluate potential emissions that could be released under various operating conditions and different chemical composition of the source water.

Indoor Environments

Led the expert team supporting the U.S. Army Criminal Investigation Division's investigation of potential environmental and building factors associated with 11 reported infant deaths in Army housing at Fort Bragg. A primary objective of the investigation was to evaluate the claim that the infant deaths were related to "Chinese Drywall" in the housing units. In addition, the investigation involved a multi-pathway assessment of potential indoor environmental contaminants including:

VOCs, aldehydes, fungi, pesticides, allergens, metals and water quality. Building factors evaluated included: mechanical systems, ventilation and building envelope performance.

Principal-in-charge of more than 2,500 indoor environmental quality assessments in large office buildings, industrial facilities, schools, hospitals, residences, and other locations.

Principal-in-charge for design and implementation of specialized IAQ and IEQ monitoring programs.

Community and building occupant/employee liaison in IEQ investigations, providing risk characterization and risk communication services.

Principal-in-charge of EH&E's study team for U.S. Environmental Protection Agency's (EPA) *Building Assessment Survey and Evaluation (BASE) Study.* Dr. McCarthy assisted EPA in developing the study design and protocols, and directed EH&E's study team in the investigation of 100 buildings over a five-year period.

Principal-in-charge of the EPA contract "Technical Support for Indoor Air Issues."

Principal-in-charge of the Centers for Disease Control and Prevention, National Institute of Occupational Safety and Health contract "Building Related Asthma in Office Buildings and Schools".

Liaison with and consultant to various local, state, and federal agencies.

Development and implementation of health and safety programs in hazardous industrial settings. Assembly and management of rapid response teams charged with the assessment of potentially toxic conditions on a variety of sites ranging from transformer fires to areas contaminated with polynuclear aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), and heavy metals, including the mapping of areas of contamination onsite and development of site monitoring programs for the remediation phase and perimeter monitoring programs at hazardous waste sites.

Development of protocols for air, soil, and surface sampling for toxic compounds.

Developed and taught IAQ professional development training programs for building owners, managers, industrial hygienists, and engineers. This work has included a training program for NASA, several *Fortune 500* companies, and a complete revision of EPA's "Orientation to Indoor Air Quality," which is presented nationwide.

Led the hazard characterization and remediation program for the disaster recovery of a major laboratory/vivarium complex that had experienced a fire. This involved detailed contamination mapping of soot and metals throughout the 400,000 square feet impacted by the fire. This data was then used to characterize risk and develop cleanup criteria.

Infectious Agents

For a major teaching hospital, led the analysis of infectious risk potential for the first OR in the U.S. to provide interventional radiologists and surgeons with immediate access to a full array of advanced imaging modalities for use during multiple procedures. The traditional placement of supply air diffusers was not feasible due to the design of the clinical equipment being installed. By using advanced computational fluid dynamic (CFD) modeling, Dr. McCarthy evaluated multiple HVAC configurations and airflow patterns for critical infection control and optimal thermal comfort. The results identified a diffuser array in a non-traditional configuration would be equally effective in preventing particles (skin cells) from impacting the surgical site when compared to conventional systems. This approach not only was subject to peer review by surgical and mechanical design teams, it also had to pass regulatory approval from the Department of Public Health, which it did successfully, before licensing.

For a large urban teaching hospital, developed an evidence based design guide for new orthopedic operating rooms based on control of airborne infectious agents. This involved evaluating the international scientific literature regarding infections from airborne particles, critically reviewing current design guides and their underlying rationale, developing a reproducible test procedure that can be used to characterize the control of potentially infectious particles, and using the testing protocol to compare the effectiveness of the various designs located at multiple institutions.

Established and led an interdisciplinary team for a major teaching hospital that was charged with reducing inadvertent exposure to environmental opportunistic pathogens (e.g., *Aspergillus, sp.* and *Legionella, sp.)* and airborne pathogens (e.g., mycobacterium tuberculosis and varicella-zoster virus). This team identified surveillance techniques, response actions, design for physical isolation of infectious individuals and developed a policy that has been successfully implemented in this 1200 bed facility.

For a major cancer treatment center, developed a method of commissioning and verifying performance of building systems and isolation facilities for state of the art bone marrow transplant facility utilizing evidence-based design and implementation of comprehensive testing procedures. After performing a design review, comprehensive testing of sub-components such as HEPA filters, evaluation of means to optimize airflow within the suites, and ensuring appropriate pressure relationships within various areas of the facility was competed and documented.

Dr. McCarthy has been the Principal in Charge of more than twenty environmental investigations regarding assessment of infections related to *Legionella* bacteria. This work has involved reviewing epidemiological data, investigating and sampling potential sources, and overseeing implementation of corrective activities. This has taken place in environments as diverse as hospitals and hotels to retail establishments.

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NTC NORTHEAST TEST CONSULTANTS

PROFESSIONAL REVIEW
for
ASBESTOS EXPOSURE

Relating to:

VICTOR A. COFFIN
v.
STATE of MAINE/DEPT. of TRANSPORTATION
CLAIM #: 8908627

Prepared for:

Katlyn M. Davidson, Esq.
Norman, Hanson & DeTroy, LLC
Two Canal Plaza
P.O. Box 4600
Portland, ME 04112

August 17, 2018

Industrial Hygiene Consultants
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NORTHEAST TEST CONSULTANTS

August 17, 2018

Katlyn M. Davidson, Esq.
Norman, Hanson & DeTroy, LLC
Two Canal Plaza
P.O. Box 4600
Portland, ME 04112

RE: Professional Review of Asbestos Exposure
Victor A. Coffin v. State of Maine DOT
NTC Job# 16562-2018

Ms. Davidson:

Please find enclosed information regarding *Northeast Test Consultants'* professional review of provided documents and other available information relating to the Worker's Compensation Claim #8908627 for benefits relating to occupational asbestos exposure claimed by Victor A. Coffin for an employment period of approximately 10-Months while working as a Bridge Operator for the State of Maine/Department of Transportation at the Carlton Bridge situated in Bath, Maine.

Mr. Coffin was diagnosed with Mesothelioma at the end of 2016.

Background - Asbestos

"Asbestos" is the generic designation for a group of naturally occurring mineral silicate fibers of the serpentine and amphibole series. These include the serpentine mineral Chrysotile (also known as 'white asbestos'), and the five amphibole minerals – Actinolite, Amosite (also known as 'brown asbestos'), Anthophyllite, Crocidolite (also known as 'blue asbestos'), and Tremolite.

The silicate tetrahedron (SiO_4) is the basic chemical unit of all silicate minerals. The number of tetrahedra in the crystal structure and how they are arranged determine how a silicate mineral is classified.

Serpentine silicates are classified as 'sheet silicates' because the tetrahedra are arranged to form sheets. Amphibole silicates are classified as 'chain silicates' because the tetrahedra are arranged to form a double chain of two rows aligned side by side. Magnesium is coordinated with the oxygen atom in serpentine silicates. In amphibole silicates, cationic elements such as aluminum, calcium, iron, magnesium, potassium, and sodium are attached to the tetrahedra. Amphiboles are distinguished from one another by their chemical composition. The chemical formulas of asbestos minerals are idealized. In natural samples, the composition varies with respect to major and trace elements. [1] [2]

The structure of silicate minerals may be fibrous or non-fibrous. The terms 'asbestos' or 'asbestiform minerals' refer only to those silicate minerals that occur in poly-filamentous bundles, and that are composed of extremely flexible fibers with a relatively small diameter and a large length. These fiber bundles have splaying ends, and the fibers are easily separated from one another. [1] [2]

Asbestos minerals with crystals that grow in two or three dimensions and that cleave into fragments, rather than breaking into fibrils, are classified as silicate minerals with a 'non-asbestiform' habit. These minerals may have the same chemical formula as the 'asbestiform' variety. [3]

Asbestos fibers tend to possess good strength properties (e.g. high tensile strength, wear and friction characteristics); flexibility (e.g. the ability to be woven); excellent thermal properties (e.g. heat stability; thermal, electrical and acoustic insulation); adsorption capacity; and, resistance to chemical, thermal and biological degradation. [1] [2]

Asbestos is typically used as a loose fibrous mixture, bonded with other materials (e.g. Portland cement, plastics and resins), or woven as a textile. [4]

The range of applications in which asbestos has been used includes: roofing, thermal and electrical insulation, cement pipe and sheets, flooring, gaskets, friction materials (e.g. brake pads and shoes), coating and compounds, plastics, textiles, paper, mastics, thread, fiber jointing, and millboard. [1] [5] [6] Certain fiber characteristics, such as length and strength, are used to determine the most appropriate application. For example, longer fibers tend to be used in the production of textiles, electrical insulation, and filters; medium-length fibers are used in the production of asbestos cement pipes and sheets, friction materials (e.g. clutch facings, brake linings), gaskets, and pipe coverings; and, short fibers are used to reinforce plastics, floor tiles, coatings and compounds, and roofing felts. [6]

Inhalation and ingestion are the primary routes of exposure to asbestos. Dermal contact is not considered a primary source, although it may lead to secondary exposure to fibers, via ingestion or inhalation. The degree of penetration in the lungs is determined by the fiber diameter, with thin fibers having the greatest potential for deep lung deposition. [6]

In indoor air (e.g. in homes, schools, and other buildings), measured concentrations of asbestos are in the range of 30 - 6000 f/m^3 (0.0003 - 0.006 f/cc). Measured concentrations vary depending on the application in which the asbestos was used (e.g. insulation versus ceiling or floor tiles), and on the condition of the asbestos-containing materials (i.e. good condition versus deteriorated and easily friable). [4]

Several factors determine how asbestos exposure affects an individual, including:

- Dose,
- Duration,

- Size, shape, and chemical makeup of the Asbestos fibers,
- Source of the exposure,
- Individual Risk Factors,
- Genetic Factors, such as having a germline mutation in *BAP1*. [7]

Although all forms of Asbestos are considered hazardous, different types of asbestos fibers may be associated with different health risks. For example, the results of several studies suggest that Amphibole Forms of Asbestos may be more harmful than Chrysotile, particularly for Mesothelioma risk, because they tend to stay in the lungs for a longer period of time. [4] [8]

Asbestos related diseases are primarily comprised of the following:

Asbestosis

Asbestosis is a chronic respiratory disease caused by prolonged exposure to asbestos. The inhaled asbestos fibers cause lung scarring and stiffness of the lungs, which prevents adequate uptake of oxygen into the blood stream.

It is difficult to distinguish from other causes of interstitial fibrosis and only confirmation of exposure to Asbestos or detection of unusually high numbers of Asbestos fibers in the lung is considered conclusive evidence of this disease.

Lung Cancer

Although a causal association between asbestos exposure and lung cancer is generally well recognized, there are still substantial controversies on how the risk might vary by exposure to different fiber types and sizes, and whether there is a risk at low levels of exposure (i.e. environmental exposures).

Particularly controversial is the question of whether Chrysotile Asbestos is less potent for the induction of lung cancer than the amphibole forms of asbestos (e.g. Crocidolite, Amosite and Tremolite), which has sometimes been referred to as the "amphibole hypothesis". [9] [10] [11]

Smoking is recognized as having a "Synergistic" effect of 50 - 100 times the risk for non-smokers also exposed to asbestos fibers.

Mesothelioma

Pleural and Peritoneal Mesotheliomas are very rare malignancies that occur in the mesothelial cells that line these cavities of the human body.

Although all forms of asbestos can cause Mesothelioma, there is considerable evidence that the potency for the induction of Mesothelioma varies by fiber type, and in particular that Chrysotile Asbestos is less potent than amphibole forms of asbestos. [12]

Asbestos is not the only cause of this disease, but it is the most important cause in modern times.

There is no definitive dose-related response information, and in rare cases has been known to occur in patients with no known occupational exposure to asbestos. [13]

Concern used to be focused on occupational exposure only, but now it is recognized that asbestos fibers are widely distributed in the general environment in many countries, cities buildings, etc. Indeed, exposure to asbestos in occupational settings has been decreasing [6], whereas this is probably not the case for non-occupational exposure. While exposure in non-occupational settings is generally much lower than in occupational settings, the levels can be not negligible, and they might be sufficient to cause disease.

Employment History

Based on provided documentation and review, the following appears to be employment/work history for Mr. Coffin:

Maine Central Railroad as the Carlton Bridge Operator from 1967 - 1968

US Navy as an aircraft mechanic for A-7 Corsairs and for Huey Helicopters from 1968 - 1971.

Maine Central Railroad as the Carlton Bridge Operator from 1971 - 1987.

State of Maine Department of Transportation as the Carlton Bridge Operator from 1987 - 1988 (10 months).

US Postal Service as a mail carrier from 1988 - 2012.

Potential Asbestos Hazards Associated with Employment/Work History

Mr. Coffin states in a questionnaire completed by him that he has Mesothelioma due to years of inhaling asbestos dusts.

Potential exposure to asbestos based on Mr. Coffin's employment/work history would be as follows:

Maine Central Railroad 1967 - 1968

Chrysotile Asbestos in Transite Wallboard, Brake Shoes and Gaskets.

US Navy 1968 – 1971

Amosite, Crocidolite, and Chrysotile Asbestos in Heat Shield, Wire Coating, Brake Shoes, and Gaskets.

Maine Central Railroad 1971 - 1987

Chrysotile Asbestos in Transite Wallboard, Brake Shoes and Gaskets.

State of Maine Department of Transportation (ME DOT) 1987 - 1988

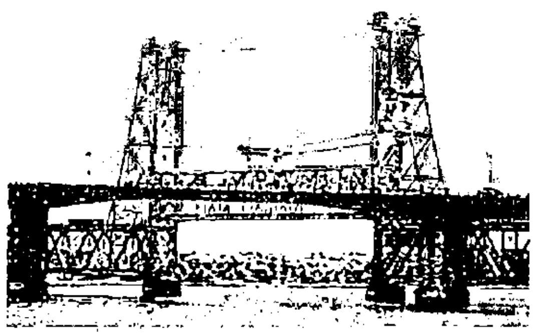
Chrysotile Asbestos in Transite Wallboard, Brake Shoes and Gaskets.

US Postal Service 1988 - 2012

Amosite and Chrysotile Asbestos in Pipe Insulations, Ceiling Tiles, Floor Tiles, Flooring Mastics, and Brake Shoe dust.

Carlton Bridge Background

The Carlton Bridge is a vertical-lift bridge built to carry one (1) railway line over the Kennebec River between Bath and Woolwich, Maine. It was completed in 1927 and shortly after a two-lane automobile platform was erected over the train track span for US Route One north and southbound traffic usage.



A vertical-lift bridge operates by raising a span section vertically by a system of motors and/or counter weights with cables and pulleys to allow for waterway traffic passage.

As part of this system, brake shoes are utilized as a secondary securing mechanism.

State of Maine/Department of Transportation Employment

OSHA Asbestos Regulations were implemented in 1971 for the control of asbestos exposure to employees. Permissible Exposure Limits (PEL's) were subsequently reduced in 1972, 1975, 1986 and 1994.

In addition, US EPA published the Asbestos Worker Protection Rule (WPR) in 1986 which covered occupational exposure to state and local municipality employees not specifically covered under the OSHA Act.

In order to impact asbestos containing materials employees would need to be properly trained in work practice and engineering controls, use of protective clothing, as well as respiratory protection usage, including medical evaluation for clearance to use respirators.

Mr. Coffin has not indicated that he was a trained asbestos worker and assigned asbestos abatement actions by the State of Maine Department of Transportation relating to perform Carlton Bridge repairs or maintenance during his 10-month employment period.

Brake shoes were not located within the Carlton Bridge Control Booth occupied by the Operator and as such the Operator was not at any significant exposure risk during operation of the bridge to airborne asbestos fiber activity.

It has been indicated that the Carlton Bridge Control Booth/Mechanical Rooms had cementitious wallboard (Transite).

No likely exposure to airborne asbestos fibers would have existed just because these transite wallboard materials were present on or within a building. Direct impact, in particular mechanical impact by dry drilling, cutting or sanding, would be required to release the bound asbestos fibers contained within the product that could then be dispersed into the air and then inhaled.

Asbestos airborne studies conducted in Australia, where a predominance of structures are constructed with cementitious wallboard materials (Transite) have indicated that in buildings comprised of these products, airborne levels of asbestos fibers are usually below 0.0005 fibers/milliliters (0.0005 f/cc), if measurable at all. [14]

US Postal Service Employment

Mr. Coffin has provided information that he was a Rural Route Letter Carrier based out of the Brunswick, Maine Post Office.

Mr. Coffin, no doubt was in and out of the building at varying timeframes during his approximately 24 years of employment.

The building, during his employment period, contained both *friable* and *non-friable* asbestos materials, and non-friable materials that could be impacted into a friable state such as floor tiles by the use of abrasive floor buffing and constant impact by such actions as rolling mail carts and dollies over the surfaces.

Friable materials can be crumbled by hand pressure and readily release asbestos fibers when impacted. Comparatively, *non-friable* materials do not crumble under hand pressure and do not readily release asbestos fibers to the surrounding atmosphere unless as previously indicated, either damaged or directly affected so as to breakdown the original state of the material.

Presence of these materials have been identified for the Brunswick Post Office location in inspection/assessment documents prepared by URS Corporation, 5 Industrial Way in Salem, New Hampshire and comprised of the following asbestos containing materials in the building:

Pipe Fittings	20%	Chrysotile Asbestos
9" x 9" Floor Tiles	5 - 10%	Chrysotile Asbestos
9" x 9" Floor Tile Mastic	3 - 8%	Chrysotile Asbestos
1' x 1' Ceiling Tiles	10%	Amosite Asbestos
2' x 4' Ceiling Tiles	10%	Amosite Asbestos
1' x 2' Floor Tiles	30-40%	Amosite Asbestos

Three (3) asbestos abatement actions were conducted at the US Post Office site in Brunswick, Maine based on available ME DEP Asbestos Project Notification database information as follows:

1. 1998: 2,240 sq. ft. of **Amosite Asbestos** Ceiling Tiles and 8 sq. ft. of **Chrysotile Asbestos** Mastic material.

2. 1999: 12 sq. ft. of **Amosite Asbestos Ceiling** Tiles.

3. 2000: 6,810 sq. ft. of **Amosite Asbestos** Ceiling Tiles.

SUMMARY of FINDINGS

It is when asbestos containing materials are damaged, disturbed or otherwise abraded that unbound asbestos fibers are released into the air and inhaled that the risk of developing an Asbestos Related Disease is increased.

The risk to health increases with the number of fibers inhaled and with frequency of exposure. Asbestos-related diseases usually take many years to develop. The risk of developing an asbestos-related disease increases in proportion to the number of asbestos fibers breathed in over a lifetime.

Significant occupational exposure to Chrysotile Asbestos may have occurred for Mr. Coffin during possible direct contact and impact of asbestos containing materials during his two (2) employment periods with Maine Central Railroad for any assigned maintenance activities. Exposure to Chrysotile Asbestos and amphibole asbestos forms (Amosite & Crocidolite) most likely occurred during his US Navy service with direct contact and impact of asbestos containing materials during aircraft and helicopter maintenance actions.

Lesser exposure appears to potentially have occurred during his 10-month employment with the ME DOT as the Carlton Bridge Operator with continuing asbestos exposure likely occurring during his 24-year employment with the US Postal Service.

It is probable that asbestos fiber contamination from vibrating ceiling tile systems could have been deposited on surfaces and materials located within the mail processing areas of the building that would then be handled by Mr. Coffin during his rural route delivery duties. [13]

Additionally, for 24-years during US Postal Service employment, Mr, Coffin would also have likely been exposed daily to airborne asbestos fibers both from the brakes of his postal truck and from other roadway vehicle traffic. [15]

<u>Based on review of Mr. Coffin's work history and the potential for asbestos exposure, it is *Northeast Test Consultants'* professional opinion that Mr. Coffin's last occupational exposure to asbestos fibers would have been during his 24-year employment for the US Postal Service.</u>

Attached is a timeline of work history illustrating exposure potential to asbestos forms and correlated to asbestos regulatory items.

Please review the provided information relating to this review and should you have any questions regarding this report, please feel free to give me a call.

Sincerely,

Stephen R. Broadhead, CIH
President

Attachment

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STATE OF MAINE

KENNEBEC, SS.

WORKERS' COMPENSATION BOARD

VICTOR A. COFFIN

vs.

STATE OF MAINE
DEPARTMENT OF TRANSPORTATION

-and/or-

STATE OF MAINE
WORKERS' COMPENSATION DIVISION

BEFORE

EVELYN KNOPF, ADMINISTRATIVE LAW JUDGE

APPEARANCES:

KEVIN M. NOONAN, ESQUIRE . . . . FOR THE EMPLOYEE

KATLYN M. DAVIDSON, ESQUIRE . . . . FOR THE EMPLOYER

HEARING HELD AT AUGUSTA, MAINE

NOVEMBER 27, 2018

I N D E X

WITNESSES	DIRECT	CROSS	RE-DIRECT	RE-CROSS
STEPHEN BROADHEAD	4	37	50	56

EXHIBITS	MARKED	OFFERED	ADMITTED
Employer 2	8	-	-
Employer 4	27	-	-
Employer 5	32	-	-
Employer 6	53	-	-
Employer 7	53	-	-

Hearing held at Augusta, Maine, November 27th, 2018 before Evelyn Knopf, Administrative Law Judge, in the case of Victor A. Coffin versus State of Maine Department of Transportation and/or State of Maine Workers' Compensation Division. Appearances are:

- Kevin M. Noonan, Esquire for the employee, Victor A. Coffin.
- Katlyn M. Davidson, Esquire for the employer, State of Maine Department of Transportation, and/or State of Maine Workers' Compensation Division.

TRANSCRIPT OF TESTIMONY

ADMINISTRATIVE LAW JUDGE: Thank you. We're back today for the second hearing in the case of Victor Coffin versus the State of Maine. It's Department of Transportation of Maine isn't it? It's DOT?

MS. DAVIDSON: Yes.

ADMINISTRATIVE LAW JUDGE: Okay.

MS. DAVIDSON: Yes, I believe so.

ADMINISTRATIVE LAW JUDGE: Okay. All right. Thank you. The parties and their representatives are the same as appeared before the Board in the September hearing

that we had here two or three months ago. We're hearing today from Stephen Broadhead, an employer witness. Will you please raise your right hand? Do you swear that the testimony you give will be the truth, the whole truth, and nothing but the truth?

MR. BROADHEAD: I do.

ADMINISTRATIVE LAW JUDGE: Thank you so much. Would you like to go ahead please?

MS. DAVIDSON: Yes.

STEPHEN BROADHEAD

After having been duly sworn, testifies as follows:

DIRECT EXAMINATION

BY MS. DAVIDSON:

Q. Good afternoon.

A. Good afternoon.

Q. Can you please state your name for the record?

A. My name is Stephen Ross Broadhead.

Q. And, Mr. Broadhead, what is your profession?

A. I'm an industrial hygiene consultant and business owner.

Q. And what is the business that you own?
A. I own Northeast Test Consultants of Westbrook.
Q. And just if you could give us a brief summary of what that business does?
A. Northeast Test Consultants is a consulting business. And the primary focus of our work over the last 30 years has been environmental consulting, which includes asbestos, lead, lead-based paint, indoor air quality, air sampling, inspection of properties for the -- for the presence of asbestos containing materials and business materials, and consulting in regard to asbestos abatement projects.
Q. And you mentioned that you are an industrial hygienist. Can you describe generally what it is that an industrial hygienist does?
A. An industrial hygienist is primarily like in occupational exposures for people in the workplace in regard to airborne pollutants -- things like ergonomics, noise, the impact of toxic materials such as asbestos -- such as lead -- airborne lead, such as heavy metals, things like silica, dust, and any -- and general safety.
Q. And can you give us a brief summary of your educational background?
A. I'm a -- an industrial chemist and an instrumental

analysis chemist. I was educated in the United Kingdom. I'm a graduate of the Royal Institute of Chemistry. And when I emigrated to America, I was employed in the utility industry; and then when I started Northeast Test Consultants, as a -- an environmental consultation. I was additionally involved in courses for the American Industrial Hygiene Association. I became a certified industrial hygienist. So my -- my business card now reads graduate of the Royal Institute of Chemistry, chartered scientist, certified industrial hygienist, and member of the Royal Society of Chemistry.

ADMINISTRATIVE LAW JUDGE: Must be rather large cards --

MR. BROADHEAD: It takes some explaining --

ADMINISTRATIVE LAW JUDGE: -- or small print.

MR. BROADHEAD: It takes some explaining, Your Honor.

BY MS. DAVIDSON:

Q. And I believe you mentioned that you were a President of Northeast Test Consultants, correct?

A. That's correct.

Q. And you're active with that business currently, correct?

A. I certainly am.
Q. Okay. And, Mr. Broadhead, in terms of familiarity with asbestos, you're familiar with this substance. Is that correct?
A. My familiarity with asbestos started in the United Kingdom in the utility industry back in 1970 when I was working in the power plants in London. And as part of a health and safety executive initiative, we were charged with collecting air samples for insulation workers who were at the time dealing asbestos. And we helped to develop the analytical methodology for determining the concentration of asbestos fibers in the air. When I came to the United States after a number of years in the U.K. industry, I worked for Central Maine Power Company as a -- initially as a chemist and then as the Deputy Plant Superintendent at Wyman Station. And I initiated their asbestos program. And then I moved on to Northeast Test Consultants and was active in the asbestos and scores -- the O'Hara Program, which started in 1987 -- and -- and being active in asbestos consulting ever since.
Q. And you were hired by my law firm in connection with this case specifically to analyze the allegation of alleged exposure at the Carleton Bridge and also to

analyze potential other sources of occupational exposure in this case. Is that correct?

A. That's correct.

Q. And in connection with being retained for that, a report was prepared providing information and analysis of the occupational exposures. Is that correct?

A. The review that we prepared -- prepared is based on documentation that we were provided with and some documentation that we were able to gather from the field -- particularly from the post office in Brunswick.

Q. And I'll show you what is marked as the Employer's Exhibit Number 2. And this is the report that was prepared with analysis and -- and findings regarding --

A. Correct.

Q. -- occupational exposure. And we'll sort of follow along in your report if we open to the beginning cover page. So you start with a section called background asbestos. And I think it -- it would be helpful with this case if you could briefly describe what asbestos is.

A. Asbestos really is a -- it's almost a generic term. It's like a family name for a class of compounds

known as silicates and particularly silicates in -- in which the crystalline structure is arranged in a particular way. There are two main groups of asbestos; one of which is the serpentine class of asbestos, which -- and is really limited to what's -- to what's called Chrysotile asbestos. And it's called serpentine because if you examine the fibers under a microscope, they appear to be very snakelike. So they classify it as serpentine. And in that type of asbestos, the silicones -- silicate structure is arranged in sheets. So they are flexible and they're able to grow in long chains. The other type of asbestos -- family member -- are what's called amphiboles. And in this case, the silicate -- for want of a better word -- monomers are arranged in straight chains. And they produce rung like structures. And typically, those would be described as Amosite asbestos, which is also known as brown asbestos; and Crocidolite, which is also known as blue asbestos; and Anthophyllite. The serpentine asbestos, Chrysotile, is often called white asbestos and it was a convenient -- a convenient way of people in identifying it, particularly when you get to things like Crocidolite, which was called blue asbestos, because that allowed the U.S. Navy to

specify it for their ships because they liked the color.

Q. And so in terms of these two main groups of asbestos and -- can you explain why the distinction of being serpentine versus amphibole would matter, especially when you're analyzing it from the context of potential occupational exposure?

A. Typically, serpentine asbestos fibers tend to be longer than amphibole fibers. They tend to be less aerodynamic, meaning that they -- they stay airborne for a longer period of time. They also -- as opposed to the amphibole type of asbestos, they tend not to penetrate as deeply into the lung tissue if they are ingested. And so typically in the literature, Chrysotile is deemed as less hazardous than Amosite or Crocidolite.

Q. And I'm sorry. Did you say that serpentine type of asbestos tends to be -- stay in the air longer or less time?

A. Longer.

Q. Okay. And I think you said earlier that serpentine cannot -- the term serpentine is sometimes used interchangeably with Chrysotile?

A. Yes.

Q. Okay. So if we -- one --

A. Chrysotile --
Q. -- they go together?
A. Chrysotile is -- is the only serpentine form of asbestos.
Q. I see. Okay.
A. Amosite, Crocidolite, Anthophyllite are all amphibole types of asbestos.
Q. And so what are --
A. So if we're talking serpentine, we're talking Chrysotile. And if we're talking Chrysotile, we're calling it white asbestos.
Q. Okay. And what about characteristics of the amphibole type of asbestos?
A. The amphibole are -- all -- all fibrous asbestos can be separated out into individual fibers, which is one of the characteristics, which makes it an extremely useful product. But it also makes it a dangerous product for human ingestion because the fibers can all be inhaled. Amphiboles -- because they are straight fibers, they can be looked at if you will as more like a dart or an arrow; whereas the serpentine type of asbestos is more like a piece of string. So when you're -- if you breathe in asbestos fibers and you're breathing in amphiboles, there is a tendency for those -- and first of all, they're very small --

some micron size. They can be ingested further into the lung tissue than a typical serpentine fiber. That's not to say that you can't get serpentine into you -- into your lung tissue, but there is a -- there's a -- a better chance that an amphibole fiber can be ingested much further down into the air where you're holding.

Q. And another term I see come up a lot in the context of asbestos is this term of friable versus not friable. And can you explain the difference between those two terms?

A. Friability really became a -- an issue in terms of human exposure because of the amount of material that was in place in industrial, and commercial, and residential structures. And simply put, there are -- the two types of product -- although they've -- there are some subcategories -- the two types of main products are non-friable asbestos containing materials -- and that would be things like Transite -- like floor tiles, exterior cladding for buildings -- and friable materials. And friable simply means that it can -- the material can be crushed or pulverized by hand pressure and create airborne dust. So the example I like to use -- because sometimes it's a little difficult to -- to kind of grasp that

as far as when you're talking about asbestos and non-asbestos -- if I go back to the kitchen and take a wooden cutting board, that's non-friable. I can cut on it. I can pick it up and bang it on the table. And I'm not going to create airborne dust. If I take a SKILSAW and cut through it, I'm going to fill the kitchen with airborne dust -- sawdust. That's a good way of thinking about non-friable materials. Friable materials are a handful of rice crackers that I want to put in my soup. I can crush those up with hand pressure and drop them in my soup. And if you look at what happens when you crush rice crackers -- especially in a dark room and you shine a flashlight -- you'll see the amount of dust that's created. So that's friability. The cutting board is not friable. Things like Transite board are non-friable. Transite is a cementitious material, which is -- in which typically it's Chrysotile asbestos -- long fibers, which act as a good binding material, mixed with a wet slurry of Portland Cement. And then they are laid out in sheets and allowed to sit and dry. And then they become a solid board, which typically needs some kind of abrasion to create dust.

Q. And what kind of materials will typically fall into

the friable asbestos?

A. Thermosystem insulation -- things like asbestos insulating block, which has a -- a fairly soft gypsum structure as filler. And that material can be -- and -- and is deemed friable, particularly if it's wet -- if it becomes wet or is damaged; materials such as ceiling tiles, which contain asbestos -- and very often those ceiling tiles contain Amosite asbestos -- the amphibole; and the ones that we've encountered over the years are usually the one-foot square ceiling tiles, which can be installed either on a grid system or using adhesive daubs and they're stuck onto the ceiling. Those are friable, particularly during removal and -- and during -- with any kind of disturbance, vibration, or -- or otherwise. And if they're water damaged, they're very friable. Other types of material that -- that -- that can be rendered friable and by hand pressure are things like insulating -- insulating gaskets, which are damaged -- something that's soft that you don't need to break up with a lot of force. Interestingly enough, floor tiles are deemed non-friable. And the issue with floor tiles is always that while they are -- the asbestos fibers are embedded in a vinyl or a rubber matrix, that matrix can be abraded by using high

speed buffer materials to clean the floors. So there are -- for a long time, we had issues with high speed buffers being used on asbestos containing floor tiles because building maintenance people didn't understand the fact that you could actually degrade that stuff by using an aggressive pad on a high speed buffer. And in fact, OSHA has now banned that practice. You can only use a low speed buffer -- and OSHA being the Occupational Safety and Health Administration.

Q. So coming back to your report on page 4, there's a section underlined employment history. And that's where you outline your understanding of Mr. Coffin's employment history up until his retirement, correct?

A. That's correct.

Q. And then moving on to the next section entitled potential asbestos hazards associated with employment -- so this section is where you identify potential sources of asbestos that Mr. Coffin may have been exposed to in connection with all of these employments, correct?

A. That's correct.

Q. Okay. And looking specifically at the employment with the State of Maine DOT from 1987 to 1988, you've identified Chrysotile asbestos in Transite wallboard, brake shoes, and gaskets, correct?

A. Yes, that's what we -- yeah, that's what we were able to identify from the documentation.
Q. And prior to coming here today to testify, you've had -- you had the opportunity to read Mr. Coffin's testimony from the prior hearing. Is that correct?
A. Yes.
Q. So you're familiar with what Mr. Coffin testified about asbestos materials he claims he was exposed to in connection with the Carleton Bridge, correct?
A. Correct.
Q. Okay. So let's talk about the wallboards -- asbestos wallboards in the three different rooms on the bridge. So can you explain -- you mentioned briefly before Transite wallboard. Can you explain what that product is and how that typically is installed -- or was installed?
A. Transite wallboard as I said before is a general -- generally, it's a mixture of asbestos fibers; and most typically Chrysotile asbestos fiber and Portland Cement -- a Portland Cement slurry, which is then mixed together and then allowed to sit as sheets of cementitious asbestos board, which -- which then are termed Transite and typically were sold in four feet by eight feet or four feet by ten feet sheets. And it was used as a structural material typically as --

as wallboard. And one of the benefits of it was that it was virtually fireproof. So for an industrial setting, you see Transite wallboard used in structures like the Carleton Bridge; you see it in pulp mills particularly -- paper mills, utilities; and you see it in residential settings where it was sold as a fire stop around woodstoves. So the -- and to create a fire -- so-called fireproof or heat proof barrier inside a room, typically the sheets of Transite would be arranged vertically with a gap -- a -- they call it an expansion gap or a vibration proof gap -- of usually about an inch or three-quarters of an inch between the edges of each sheet, so that they didn't rub together. And that was less of I think a concern about creating fiber dust; as -- more as a -- just creating a -- an abrasion issue when these were originally installed. And then over the top of that gap was usually placed a -- a cover section, which was probably an inch and a half -- could be two inches wide. And that allowed each sheet to move independently of the others. Generally speaking in -- in my experience, the cover strip had enough clearance, so that these three components would move independently of each other.

Q. And you've read Mr. Coffin's testimony describing the

wallboard and the rooms on the bridge, correct? You recall that testimony?

A. Yes.

Q. And at one point, he talked about particularly when trains were passing through creating vibration and seeing the wallboards move.

A. That's not untypical.

Q. And is this description of how he -- he saw the boards moving consistent with your understanding of how wallboards are typically installed?

A. The way that the -- Mr. Coffin's testimony described the movement and what are described on the -- what I've seen as typical construction, that's consistent with the design.

Q. And in terms of the likelihood of the wallboard rubbing against another piece of wallboard and creating dust, what was the likelihood of that happening?

A. There is some likelihood. It's -- I think it's fairly minimal.

Q. And you've read about Mr. Coffin's testimony with respect to there being dust in these -- in the various rooms, correct?

A. Absolutely, yeah.

Q. And -- well, let me ask you this: If -- if an

individual is just to look at dust, can an individual visually identify asbestos dust from other forms of dust?

A. No.

Q. How would you be able to determine asbestos dust?

A. The -- there are a number of analytical methods around to determine presence of asbestos fiber in dust. Visually, it's not possible because we do not have that resolution without the aid of microscopes, or electron microscopes, or transmission electron microscopy. So visually, you might see dust. You can't -- and you might see fibers. If you can see fibers or clumps of material, you still can't tell whether it's asbestos, paper, or other miscellaneous materials. There's no way to do that. So the only way then is to take samples and then analyze them using microscopy techniques.

Q. And in an environment such as the Carleton Bridge, would you expect there to be dust from other sources?

A. I'd be amazed if there was not, so -- whenever you've got -- well, we'll call this a semi-industrial type of environment because you've got -- you've got a railway system, you've got a -- you've got large equipment, you've got cables, you've got road traffic, you've got combustion sources -- combustion

engine sources, you've got -- say you've got particulates -- general particulate, you've got road particulate, which could be silica or silicates -- non-fibrous -- you've got unburned carbon, which can create -- does create a lot of first airborne material and then settles out as dust, and you've got just general dust -- things like loose particles of rubber from tires, which stripped off because of the road transit. You've got road surface materials, which are again stripped off because of road traffic. So there are all kinds of components in -- in regular dust.

Q. And so based on your understanding of Mr. Coffin's testimony and the -- you know, environmental circumstances of the bridge, how likely is it do you think that there would have been exposure from the wallboard -- asbestos in the wallboards?

A. I think it's unlikely.

Q. What typically conditions would have to be present for someone to be exposed to asbestos from wallboard?

A. There would have had to have been some kind of mechanism that physically damaged the wallboard. And that would -- that would be where somebody has either drilled through the wallboard, used -- used power saws, or impacted the wallboard with some kind of

tool.
Q. And is -- is wallboard typically painted when it's installed?
A. In my experience, usually it -- there's -- the -- at least the interior surfaces are usually painted. And where I -- where I've seen Transite board in paper companies, hospitals, utilities, they love to paint. It removes the limited -- if it doesn't paint it -- you know, the old adage from the -- from the services. So -- and it's a good way of -- of mitigating any surface dust that might be on the -- on the wallboard. It's a cheap way of dealing with it.
Q. Now turning to the -- the -- the brakes and potential asbestos from the brakes, what type of asbestos is typically associated with brakes?
A. Typically, it was Chrysotile asbestos, serpentine asbestos. Although you've -- in manufacturing, all asbestos containing materials -- there was usually some kind of cross contamination. So you may get -- you may get some small amount of amphibole -- you know, Amosite, and even Crocidolite in some brake shoes. But typically, they were used in Chrysotile.
Q. Would that be friable or non-friable material?
A. In a brake shoe, it would be non-friable. Obviously,

it could be rendered friable.
Q. And with respect to just the Carleton Bridge in general, what's your understanding in terms of the type of bridge that this bridge was?
A. My understanding of this was a -- a centered section lift bridge -- a vertical lift bridge designed using a series of cables and electric motors to detach the center section between two gantries and raise or lower the roadway or the rail -- rail bed to allow the passage of ships underneath.
Q. And what did that mean in terms of operation of the braking system?
A. If you look at this type of bridge, what you've effectively got are two travel terrains, which have been raised vertically, and through cables, and pulleys, and the motors. Those are allowed to raise and lower the bridge in controlling -- typically controlling speeds by use of the -- of the motors. Once the bridge -- the center section is up or down -- usually up -- then a brake -- the braking system is applied as a secondary safety device.
Q. What does that mean?
A. Well, it -- my -- my understanding and my background with the utilities is -- and particularly with travel trains and this type of arrangement -- is that the

speed of the -- of the cable release and speed of the travel of the bridge is controlled by the electric motors. The -- holding a bridge in place -- that's where the brake -- the brake units come into play. So when it's -- when it's raised, you're using the brakes as a secondary safety device. So you're not trying to slow down the bridge with brake shoes like you do with a vehicle. So it's -- it's more -- like if you're using engine braking in a truck, you're not really using the brakes. You're using the engine to brake the truck, so to lower and control its speed. But in cars -- in cars and trucks, the braking system is the primary mechanism -- mechanism to slow the truck down. The engine can be a secondary. In this type of arrangement, the engine is -- is basically the primary and then the -- the brake -- the brake system is the secondary. That's my understanding of the way this -- these bridges work.

Q. Mr. Coffin had testified about there being an incident that he responded to in this engine room, where the braking system is located, and entering when there's a lot of black smoke. Do you recall reading that testimony?

A. I do, yeah.

Q. And what are your thoughts on the description of that

incident with him for potential asbestos exposure?

A. In terms -- in terms of relative exposure, I think the asbestos issue is fairly minor in terms of occupational exposure to contaminants. The black smoke indicates that there was a -- a high concentration of unburned carbon in the air. That's typical for low temperature fires or combustion where you've got materials like grease, polymers, rubber that are -- that are -- caught fire or are getting close to combustion. And the -- the smoke is really a fairly dense particulate and it's very black. So if you think about what a -- if you set a tire on fire, you get a very aluminous flame. You get clouds of black smoke. You've got the same kind of issue. There's a lot of unburned carbon up in the air.

Q. So does the -- the -- that description of that incident lead you to believe there was an issue with the -- any asbestos in the brakes for potential exposure in response to that incident?

A. It's possible that you may get some asbestos release from them. But to get a significant exposure, I think you'd have to completely strip out the -- the brake shoes and render all of that material friable. I think that's unlikely given the -- given the description. Being in that room when it was filled

with black smoke -- Mr. Coffin was lucky to get out of there. In fact in -- in most fire situations, it's fairly well documented that smoke inhalation is what kills most people before they -- they succumb to the fire.

Q. Now turning to Mr. Coffin's employment after he left the State of Maine and the Carleton Bridge, Mr. Coffin went to work for the United States Postal Service based out of the Brunswick office. And you have had the opportunity to review documents that were received from the Postal Service regarding the presence of asbestos at that facility, correct?

A. Correct.

Q. And can you explain to me sort of your analysis and what you found noteworthy about the presence of asbestos in potential post office?

A. That was an interesting facility. The asbestos containing building materials -- ACBM's as they are referred to in many reports and including these survey reports -- were fairly ubiquitous through the building in terms of thermal system insulation, piping insulation, pipe-fittings -- which wouldn't by definition be termed friable. So those -- if they were damaged, they would be -- in terms of -- you know, significantly damaged or -- or damaged with the

ability to release airborne fiber, they would -- and any debris would be friable. You could pick it up and crumble it. Floor tile -- nine by nine asbestos containing floor tile -- and significantly we've seen floor tiles with two or three percent asbestos. The analytical results of the floor tiles in the Brunswick Post Office had over ten percent in cases. So that's a fairly high concentration of asbestos containing material in a floor tile. So that material was present throughout. And -- and that material, as I said before, can be abraded by foot traffic, by rolling carts across it, and by sand and grit, by floor-cleaning methods. So those methodologies can degrade the floor tile and they can release asbestos in -- as an asbestos dust. And we've seen Chrysotile and we've seen Amosite as asbestos containing materials in those floor tiles. Significantly, they were also one-by-one -- twelve inch by twelve inch asbestos -- asbestos containing ceiling tiles throughout the building. And the -- the survey materials that I saw also included some photographs. And in those photographs, it's apparent that there were damaged and dislodged ceiling tiles in various parts of the building. And the ceiling tiles are also subject to a -- a couple of different

abatement actions by contractors.

Q. If I could briefly stop you there? So you mentioned a photograph. Is this the photograph that you looked at?

A. Yeah, that's --

MS. DAVIDSON: This is --

MR. BROADHEAD: -- that's photograph 1.

MS. DAVIDSON: -- Exhibit Number 4.

ADMINISTRATIVE LAW JUDGE: Thank you.

MS. DAVIDSON: Yeah.

MR. BROADHEAD: I think the one I've got is actually colored, which I think makes it --

MS. DAVIDSON: And I apologize, Judge Knopf. I should have, based on these page numbers --

ADMINISTRATIVE LAW JUDGE: Oh, I --

MR. BROADHEAD: Yeah, if you want, I have better photograph than yours.

BY MS. DAVIDSON:

Q. Do you want to show us what you're looking at?

A. I'm just looking at this one.

Q. Okay.

A. It's a little clearer.

Q. A little clearer?

A. Yeah, but same --

Q. So --
A. -- basically the same -- same idea.
Q. So, Mr. Broadhead, why don't you explain to us what this top photograph -- your interpretation of that?
A. This photograph depicts -- this the vertical wall and this is the part of the ceiling tile system. And it's hard to tell on this photograph whether or not this was a splined system where the edges of the ceiling tile are grooved and they fit into a spline system almost like this, so that they're held up by a -- a spline slightly similar to that, but a little bit different because the splines are -- are hidden.

ADMINISTRATIVE LAW JUDGE: Ah. I'm just going to say for the record that I was pointing to the ceiling --

MR. BROADHEAD: Yeah.

ADMINISTRATIVE LAW JUDGE: -- in this room.

MR. BROADHEAD: Yeah. Well --

ADMINISTRATIVE LAW JUDGE: This is suspended, so we've got some frame of reference.

MR. BROADHEAD: We've also found -- although not in the Brunswick Post Office -- these types of ceiling tiles also contain asbestos sometimes.

ADMINISTRATIVE LAW JUDGE: Hmm.

MS. DAVIDSON: Oh, joy.

MR. BROADHEAD: So these ceiling tiles are definitely subject to vibration and dislodging, as you can see in this photograph. One of them is actually dislodged and is on -- on the verge of falling out. These ceiling tiles I believe contained Amosite. And I'll be glad to look for that. That was my understanding and I think that's true. Yeah, these -- if I -- not only did the one-by-one ceiling tiles contain ten percent Amosite, but also the two-by-four -- two feet by four feet -- contained Amosite asbestos. And those could be of this -- this type -- the drop --

ADMINISTRATIVE LAW JUDGE: The type in this room?

MR. BROADHEAD: -- the drop in ceiling tile.

ADMINISTRATIVE LAW JUDGE: Okay.

MR. BROADHEAD: Again, those are also friable. And those are also known to create airborne fiber and airborne dust.

BY MS. DAVIDSON:

Q. And what can cause -- you mentioned vibration. What can cause these ceiling tiles to vibrate?

A. Interestingly enough, traffic going by the building -- heavy traffic in particular. But more significantly, you can also get vibration through the

air movement, through air erosion, from HVAC systems, and also through changes in pressure -- differential pressure from one room to the next. So if you've ever gone into a room where you open the door and the ceiling tiles seem to flip, that's a -- a significant source of vibration. And maintenance activities are -- are often cause for -- not vibration, but certainly dislodgement -- water damage, ceiling/roof leaks. Some of these ceiling tiles, particularly these drop panels and the -- in these, not only contain asbestos, but some contain mineral fiber, as well -- plus fillers such as a gypsum filler or -- or some other kind of manmade mineral fiber and -- and a -- an adhesive, which holds everything together. One of the problems with that is when they get wet, they create this type of staining, which again interferes with the structural integrity, and they create a fermentation. And the -- the produce of fermentation is butyric acid, which is the principal ingredient of baby puke. So if you've ever smelled that -- that's why these things smell so bad and they degrade it, so --

Q. And you mentioned water damage. And on the same page with that photograph, what's -- what does that photo below indicate to you?

A. I believe this was taken in the basement. And this is a -- it's -- in terms of a plaster wall that has lead paint -- based paint peeling off it. And to me, this indicates that there is -- there's either water damage or condensation coming through the wall. And this is what can be called latens [PHONETIC]. It's the salts, which are inherent in either concrete or plaster wall. And what happens -- because of differential pressure across the wall, you get the salts bleeding out and they create -- they set up basically salt scabs, which then peels off the surface layer of the plaster or the -- or the cement and peels off any paint, as well. So typically, we see roof leaks in buildings -- and roof leaks -- this type of stuff subgrade where you've got moisture coming through. We see water coming up through concrete slabs that have incorrectly applied or no moisture barrier applied. So there's all kinds of -- have a use for -- for leaks as you can tell -- fire protection systems that have either caused condensation, and then leaked, and then dropped the ceiling tile into the room or maintenance that removed it and left the fiberglass insulation or other insulation bare for your enjoyment.

Q. And these ceiling tiles that contain Amosite -- are

they considered to be friable material?
A. Yes, they are.
Q. Okay. And when you mentioned vibration causing airborne dust, how likely when that occurs is it that asbestos fibers are being released into the air?
A. Airborne dust by definition is dust or fiber in this case. It's highly likely that you've got Amosite asbestos in the air released in -- in whatever quantities from these -- from these ceiling tiles.
Q. And I believe you mentioned in your testimony about abatement projects being done at the post office.
A. Yes. We had -- in the documentation that I reviewed, there were several asbestos abatement projects that were undertaken inside the Brunswick Post Office.
Q. And I'll show you what's been marked as the Employer's Exhibit Number 5. Are these the documents you're referring to?
A. These are the Department of Environmental Protection database notification files. Whenever an abatement is undertaken of a particular size -- anything over three -- three linear or three square feet of friable material -- it's required that the DEP is notified ten days prior to the project. And that -- that notification is usually done by the contractor. And the DEP maintains a -- maintains a database that not

only contractors, but consultants like ourselves, and projects.

Q. So these three pages of documents from the DEP are documenting abatement projects that were done at the U.S. Postal Service in Brunswick, correct?

A. Yeah, at different times. One is October 1998, one is December 1999, and one is May 2000.

Q. What can you tell about the materials that were removed?

A. Some of this is ceiling tile, which again amounts to -- they don't actually specify whether or not it was the twelve-by-twelve or the two-by-four -- but asbestos containing ceiling tiles. Some of this was mastic. And again, the second one was ceiling tiles.

Q. And what is mastic?

A. Mastic is a general term for -- within the bounds of the asbestos industry, mastic is a general term for organically bound asbestos materials used as glues, used as bonding materials, used as -- as roofing cement. All those are mastics. And typically, they do not release airborne fiber. And to a large extent, they are now excluded from many of the engineering controls that friable materials require unless you render the mastic friable. And you would do that by using things like shotblasting or abrasing

machines. So you can -- you can deal that -- deal with those with solvent chemicals and it's -- those are not then rendered friable.

Q. And with respect to Mr. Coffin's employment at the postal service, he testified that he was in that building daily to begin his day to sort and collect the mail for his route; and at the conclusion of his day to return the mail that was outgoing or undeliverable mail. He also testified that he spent every working day just about out on his delivery route to deliver mail. And in terms of his delivery route, do you see there being any probable source of exposure from doing that particular activity?

A. Well, the -- the potential for asbestos exposure from vehicle -- again, brake pads, from road materials, from just being out and about on the -- in a -- what I would call non-country type of area. You're not in the middle of a desert. You're in a developed country. We're all exposed to some level of asbestos. We're all -- we've all been exposed to some level of asbestos. If you're out on the road a lot, if you're in and out of a vehicle, if you drive -- if you drive with the windows open, yeah, you're going to be exposed to some level of -- of asbestos. Did I misunderstand your question? Were you asking

me about exposure inside the post office or outside the post office?

Q. I was asking about outside --

A. I'm sorry.

Q. -- on the delivery route.

A. Yeah, I think it's --

Q. Um hmm.

A. -- I think it's unarguable that -- that you would not be exposed to some level of -- of asbestos --

Q. And then --

A. -- along with all the other particulates that we are all exposed to.

Q. And so, Mr. Broadhead, after reviewing all of these documents, did you come to a conclusion about -- or come to have an opinion about where Mr. Coffin was likely last exposed to asbestos in an occupational setting?

A. Well, I looked at the exposures -- potential exposures that we could -- could examine throughout his employment as -- as described by the documentation. When I looked at the -- the amount of asbestos that was inside the Brunswick Post Office, and how -- what the distribution of that material was, and what the condition of the material was, and the fact that there were abatement projects --

although we've been unable to unearth any airborne fiber monitoring that should have been done by any of those contractors as part of the abatement procedures -- either documentations being lost or nobody can remember doing any kind of air sampling, which they should have done. When I look at the distribution of those materials, the types of materials, the friability of the ceiling tiles, and the condition of the ceiling tiles, the fact that floor tiles had been removed at some point because mastic had been exposed and that was then abated as part of these projects. So how -- how those ceiling -- those floor tiles were removed nobody knows. Were they removed in a friable manner or a non-friable manner? We -- we can't assess that. When I look at -- at the amount of material, the distribution, when I look at the amount of time Mr. Coffin had to have spent inside the building on a daily basis over a 20 year period, my conclusion is that he was exposed to some asbestos -- airborne asbestos at the post office in Brunswick. I think that's inescapable.

MS. DAVIDSON: Thank you. Those are all my questions.

ADMINISTRATIVE LAW JUDGE: Okay. We're going to take a little bit of a break --

MR. NOONAN: That's great.

ADMINISTRATIVE LAW JUDGE: -- and then we'll come right back. Okay?

MR. NOONAN: Um hmm. Thank you.

OFF THE RECORD

ADMINISTRATIVE LAW JUDGE: You're still going to be under oath. Okay?

MR. BROADHEAD: Yes.

CROSS EXAMINATION

BY MR. NOONAN:

Q. Sir --

ADMINISTRATIVE LAW JUDGE: Hold on just a second.

MR. NOONAN: Oh, I'm sorry.

ADMINISTRATIVE LAW JUDGE: Hold on just a second. Are we ready now? It takes a second to get on. Go ahead.

BY MR. NOONAN:

Q. Mr. Broadhead, I don't plan to go into your report in a lot of detail and I thank you for taking the time

to testify today. I just wanted to ask you a few questions. With regard to the asbestos, you spent a lot of time distinguishing between Amosite and Chrysotile asbestos, and the relative dangers of each. Is that --

A. Correct.

Q. -- fair to say?

A. Yeah.

Q. Would you agree with me that all forms of asbestos are hazardous?

A. Yes.

Q. And all forms of asbestos can cause cancer?

A. Yes, that's correct.

Q. All forms of asbestos can cause mesothelioma?

A. That is correct.

Q. And meso -- the most likely cause of mesothelioma is asbestos exposure?

A. It's the only cause.

Q. The -- it's a little unclear from your testimony. Have you ever actually spent any time on the Carleton Bridge besides driving over it perhaps?

A. No.

Q. Did you ever spend any time in the control rooms?

A. No.

Q. Did you ever spend any time in the machinery spaces?

A. No, I did not.
Q. Were you personally involved in any of the asbestos abatement on the bridge?
A. No.
Q. Do we have records of the asbestos abatement on the Carleton Bridge?
A. We have a --

MS. DAVIDSON: Kevin, are you asking generally or just Mr. Broadhead specifically if he's seen the documents?

MR. NOONAN: Yes. Yes, if he's seen the documents.

MS. DAVIDSON: Okay.

MR. BROADHEAD: Yes, I did see a document. And I believe it was RJ Enterprises who did the removal. I don't think I've got that with me.

BY MR. NOONAN:

Q. Well, I guess part of the reason I ask, it seems like we have a fair amount more documentation about the asbestos abatement at the post office than we do at the bridge. Would you agree with that?
A. In -- in terms of nullifications from the DEP?
Q. Yes.
A. Yeah, I've got copies of those and I have seen a

notice of an abatement at the bridge. Oh --
Q. Do you know why we don't have comparable information about the bridge?
A. I don't have it with me. That's --
Q. And is it fair to say as a general principal, the more you're exposed to asbestos, the more likely you are to develop an asbestos related disease?

MS. DAVIDSON: Objection; foundation. I think this is going into medical territory.

ADMINISTRATIVE LAW JUDGE: I suspect Mr. Broadhead could tell us that himself.

MS. DAVIDSON: Okay.

MR. BROADHEAD: There is -- certainly in the literature, there is certainly a dose response relationship.

BY MR. NOONAN:

Q. And -- and there are a lot of -- I'm sure there's a lot of other factors that -- the type of asbestos, the -- the condition of the asbestos, the general environment, and things like that -- genetics even?
A. Yeah, so concentration of airborne asbestos, the type of airborne asbestos -- Crocidolite particularly being recognized as having a higher incidence of asbestos related disease -- duration -- so long --

even low -- low concentrations, but for a long period of time can be significant. So when we talk about dose response, we're not only talking about a short-term high concentration. We can be talking about low -- low concentrations for an extended number of years.

Q. We don't have any air quality testing from either the post office or the Carleton Bridge?

A. Not that I've found, no.

Q. And -- and that would be helpful information to have to determine the asbestos exposure of Mr. Coffin or anyone who was working in those places?

A. It would be helpful, yeah.

Q. So your conclusions are -- are based somewhat on speculation without that type of information?

A. They are -- my conclusions are based on -- as I said, I think reasonable -- if you want to use speculation or assumption -- that if you've got widespread distribution of materials, much of it appears to be in poor condition and much of it appears to be friable as opposed to -- that's at the post office -- as opposed to the Carleton Bridge, which was primarily my understanding is non-friable materials affixed to the walls, then I think it -- I think that's a reasonable assumption.

Q. What is your understanding based upon regarding the asbestos at the Carleton Bridge?
A. The descriptions that I've seen, and Mr. Coffin's testimony, and --
Q. Well --
A. -- the materials that were described in the abatement report, which I -- I -- I may have in my briefcase. I don't have it here.
Q. You wrote your report before Mr. Coffin ever testified. So you didn't have that when you concluded what asbestos was at the Carleton Bridge?
A. That's -- that's true, but I -- I did have the abatement report.
Q. Your testimony with regard to the wallboard at the Carleton Bridge --
A. Um hmm.
Q. -- and how it is installed -- that's not based upon any specific knowledge of the wallboard at the Carleton Bridge. Is that correct?
A. That's based on what I've seen in -- for a typical installation of the materials. And when I reviewed Mr. Coffin's testimony -- in fact, he actually describes the -- the similar type of construction.
Q. Well, he describes walls moving when exposed to significant vibration, correct?

A. He actually describes the -- the construction, as well. If you look on -- yeah, if you look on page 16 -- if I can refer you to that -- there was testimony -- he described basically the same thing that I've seen. It wasn't a -- it wasn't a one-piece wall. It was sections. And these sections were moving independently of each other. And there's a -- a panel between -- a strip between each panel -- a small strip just that I can see -- probably about an inch.

Q. Well, it's correct he testified that the walls were moving when exposed to vibration?

A. That's what he -- yeah, that's his testimony. The --

Q. And --

A. The construction is consistent with what I described.

Q. Sure. And he testified that when the walls -- walls moved, dust would be created?

A. That was -- that's his testimony, yes.

Q. Okay. You have never been there?

A. Correct.

Q. You've never spoken to anyone who's been there?

A. Correct.

Q. You would agree that there was a tremendous amount of vibration involved in a facility such as that?

A. I wouldn't disagree with that.

Q. Right. I mean you -- you talked about the fact that at the Brunswick Post Office cars going by on the street --
A. No, I said heavy traffic.
Q. Okay.
A. Yeah, sorry.
Q. Heavy traffic going by the street --
A. Yeah.
Q. -- of a building might cause vibration. Well, I assume it's even more likely to have vibration when you have tractor trailer trucks and trains going across the bridge?
A. Absolutely, yeah.
Q. So if you can be exposed to vibration at the post office from heavy traffic going by on the street, he's exposed to even more vibration on that bridge?
A. That's true.
Q. And --
A. And the -- the difference that I was referring to in terms of airborne fiber or exposure is the materials on the bridge were not friable. They were not intrinsically friable. The -- and the ceiling tiles in the post office were -- are friable -- or were friable materials.
Q. But friable only --

A. So that -- that was my distinction.
Q. But friable only means that it can be crushed with your hand?
A. Well, that's a significant difference.
Q. Yeah. Well, constant movement can wear down even non-friable material can it not?
A. It can if they're -- if they're in close proximity.
Q. Um hmm.
A. Usually, the way that -- as I described, the way those panels are installed, they leave a gap so that you don't get constant rubbing.
Q. So if -- if Mr. Coffin described that these walls are moving, the trains are going across the -- the bridge, the place -- the whole place is shaking, the walls are moving, and there's dust created, he couldn't be right about that?
A. He could be right about that, absolutely.
Q. And if there was dust created, it would be asbestos dust?
A. Not necessarily. It could be all kinds of dust that would be dislodged. These are not hermetically sealed rooms as far as I know. That's not -- that's not my experience of those types of enclosures.
Q. Sure. But when walls are moving and dust is created, isn't it a fair assumption that that's where the dust

is coming from if that's what you're observing? I mean if I sit here and tear up these papers over the course of the next half hour and I get paper dust on my pants, even though I haven't analyzed it, it's fair to say that's probably paper dust is it not or --

A. Yeah, that's a reasonable -- a reasonable idea.

Q. With regard to the brakes, I'm also curious about -- again, you testified that the braking that -- first of all, you're not a bridge engineer?

A. Absolutely not.

Q. All right. And you're not testifying as a bridge engineer?

A. No, I'm not.

Q. And you've never been to the Carleton Bridge to see how the bridge is raised and lowered?

A. That's true.

Q. And you're not suggesting are you that the bridge is raised and lowered without using the brakes?

A. No. What I'm suggesting is that the motors and the cable system are the principal mechanism controlling the speed on that type of bridge so far as I know. But as -- I'm not testifying as a bridge engineer.

Q. Sure. Would you agree that every time that span is raised and lowered, the brakes are used?

A. To some extent, yeah.
Q. Would you agree in the wintertime they're even used even more?
A. Possibly, yeah.
Q. And these -- these are not brake pads like one on -- on one's vehicle. These are much larger and more industrial --
A. Yeah, these are industrial sized brakes.
Q. And the brakes are not in -- in -- when I'm driving around out in the -- in the free countryside air. They -- they're actually in an enclosed space?
A. Yes, correct.
Q. All right. So if -- if Mr. Coffin can be exposed to asbestos driving around the countryside on his rural route, he would be substantially more exposed to brake pad dust in an enclosed machinery space on the Carleton Bridge?
A. If he spent extensive periods of time in that space, possibly.
Q. Okay. You didn't mention that as one of the potential causes that was asbestos exposure?
A. I -- I did not mention that?
Q. That -- that his exposed -- exposure to asbestos brake pads is -- is one of the -- his potential exposures while working at the Carleton Bridge?

MS. DAVIDSON: Objection. I think it misstates the testimony and the report.

ADMINISTRATIVE LAW JUDGE: Perhaps we could get some clarification?

BY MR. NOONAN:

Q. Well, you would agree that -- that is one of his exposures?

A. A potential exposure.

Q. Yeah. I mean you do -- I mean -- I -- I -- as I understand it, you do agree he was exposed to asbestos on the Carleton Bridge?

A. To some extent, yeah.

Q. So your entire knowledge of the asbestos on the Carleton Bridge is based upon that one abatement report and --

MS. DAVIDSON: Objection. Again, I believe that misstates the testimony.

BY MR. NOONAN:

Q. When you wrote your report, your entire basis for your knowledge of asbestos exposure on the Carleton Bridge was that one abatement report?

A. That's true, yes.

Q. Okay.

A. And -- yes, I -- that's true.
Q. And -- and what is -- identify that report for us.
A. It's a -- an abatement report, which includes the materials -- in fact, it includes the specifications I think for the abatement removal. And there was a disposal receipt as I recall.
Q. What is the general latency period from exposure to asbestos to the development of mesothelioma?
A. Typically, it's somewhere between 30 to 40 years.
Q. What's the earliest exposure to development of mesothelioma?
A. The -- I mean the literature shows -- really shows -- I think the earliest that I've seen is 20 years.
Q. In other words, just -- I mean if I'm exposed -- I could swim in a bath of asbestos today. I'm not going to develop mesothelioma tomorrow, or next week, or next month, or probably even next year. It's something that develops long after the exposure has taken place?
A. In the general population, that is true. I can't say that -- and again, I can't testify as a medical expert, so I can't say that there aren't cases in the literature -- in the literature because I believe there are. And as you -- as you mentioned, there are some genetic predispositions to the development of

mesothelioma when exposed to -- to asbestosis and --

Q. You --

A. -- there -- there have been cases -- although I don't have the references with me -- of young -- young people being exposed to incidental asbestos from say an asbestos worker's clothes. And they've developed mesothelioma prior to being over 40 years old. So this is -- when you're talking about latency periods, you're really talking in general -- generalities about the general healthy population. So if you're immunocompromised, if you've got a genetic disposition, then all bets are off in terms of what really -- what latency periods might be -- might be applicable to hopefully people in the general population.

MR. NOONAN: Thank you. I don't have any other questions.

ADMINISTRATIVE LAW JUDGE: All right. Thank you. Anything else?

MS. DAVIDSON: Yeah.

RE-DIRECT EXAMINATION

BY MS. DAVIDSON:

Q. I just want to clarify, Mr. Broadhead, you -- you

were asked about vibration and you testified about the relevance of vibration regarding releasing dust from the ceiling tiles at the Postal Service, and then also asked about vibration on the bridge causing the potential release of dust from the wallboard. And why -- can you explain the distinction?

A. The -- the ceiling tiles that we've got photographic evidence of, and the fact that they were abated, and the fact that they are over ten percent Amosite material first -- and they are friable material, and they're recognized as friable material gives me concern about -- and vibration and damage in that facility in not only the airborne fiber, but where the settled fiber ends up. And if you look at the distribution of where those ceiling tiles were, they were throughout the building. So if you were -- for example, turn up your paper and you found dust on your trousers in the post office, you couldn't definitively say was it paper dust or did you get it from the ceiling tiles because -- you know, as you can see -- look behind you. Ceiling tiles -- when they come out of -- when they're damaged, they fall out. Particles -- airborne particulate can end up settling out. So with that issue, in the Carleton Bridge as -- as I understand it and I think I can --

from what I can ascertain, that there was Transite wallboard material, which was affixed to the walls -- and when I read Mr. Coffin's testimony, it's -- is consistent with what I've seen for construction and use of non-friable Transite wallboard that was affixed to the wall. And even though that was moving, the Transite was not degrading in high concentrations to release airborne fiber. Now you might have got some visible dust. But the visible dust dropping out doesn't mean that the airborne fiber concentrations, which you can't actually see, are significant. Without airborne fiber data, the best I can do is say this is non-friable material. I don't see a mechanism for abrasion occurring because you've got transient -- transient strips, you've got separation strips, and it's a non -- and as we said before, it's non-friable compared to the friability of ceiling tiles. So I -- I don't know if that answers your question or not.

Q. And you were asked about what documents you relied upon in terms of asbestos and/or abatement done at the Carleton Bridge.

A. Right.

Q. And I'll show you these documents that are already in evidence as -- I believe this one's -- I think --

this -- this one is in evidence as Employer's Exhibit Number 7.

A. Okay. This is the New Meadows abatement documentation that I was referring to.

Q. And you had those documents to review --

A. Correct.

Q. -- and analyze before preparing the written report?

A. Yes.

Q. And I'll also show you documents that came from the State of Maine that's in evidence as Employer's Exhibit Number 6.

Q. Yeah, these are -- these are the disposal receipts for asbestos containing waste materials from the Carleton Bridge. And they were provided from New Meadows Abatement to Brian Watson of the Cianbro Corporation. And they describe the materials that were taken out as non-friable wrapped materials from the Carleton Bridge, and then the -- this -- a number of those are receipts. And this is the bidding section from -- the bid documents that -- this is dated April 6th, 2000. And this is the special provision section 104 from the bid documents I believe for the demolition of the bridge. And it says that lead and asbestos abatement are required on this project. And the -- they describe the existing

control house containing asbestos. And then this is section 815, which says asbestos removal, including Transite panels, floorboards, sheeting, and insulation is included in all of this item.

Q. And these are documents you reviewed --

A. Yes.

Q. -- in connection with preparing your report, correct?

A. Yes.

Q. And you also had some discovery information that was provided by the employee that you relied on when making certain assumptions about the presence of asbestos, correct?

A. Correct.

Q. And you were asked about on the Carleton Bridge the brakes being in an enclosed space and the likelihood of exposure being present when the brakes were in operation. And is -- exposure in that situation would really only happen if the individual was in the room that the brakes are housed in at -- at the time they're in operation. Is that correct?

MR. NOONAN: Objection; leading.

ADMINISTRATIVE LAW JUDGE: Yeah, seriously.

BY MS. DAVIDSON:

Q. What's the likelihood of an individual being exposed

to brakes on the Carleton Bridge if they're not present in the room during operation?

ADMINISTRATIVE LAW JUDGE: Can you just say that again? I'm sorry.

BY MS. DAVIDSON:

Q. What's --

MS. DAVIDSON: If I can remember correctly.

ADMINISTRATIVE LAW JUDGE: Yeah.

BY MS. DAVIDSON:

Q. What's the likelihood of an individual being exposed to asbestos from the brakes on the --

ADMINISTRATIVE LAW JUDGE: There you go.

BY MS. DAVIDSON:

Q. -- Carleton Bridge when they're not in the room while in operation?

A. I -- I think it's unlikely that you would be exposed to significant concentrations, although you could be exposed to whole concentrations of asbestos if the brakes were -- were shown to be -- being rendered -- the brake pads were shown to be rendered friable and wind -- you know, higher -- you know, wind direction was able to move the air from one place to another

where you've got an employee. But you need a lot of conditions --
Q. Um hmm. And --
A. -- to -- to line up.
Q. And in terms of -- we talked -- or you've testified and were asked questions about potential exposure to asbestos while working at the Carleton Bridge. And if there is some exposure from that employment, does that change your opinion about the likelihood of exposure while working at the postal service?
A. No.
Q. Thank you.
A. No.

MS. DAVIDSON: Those are all my questions.

ADMINISTRATIVE LAW JUDGE: All right. Thank you. Anything else?

RE-CROSS EXAMINATION

BY MR. NOONAN:

Q. With regard to the brake pads again, I mean the brake pads on the bridge mechanism are much larger than the brake pads on a vehicle?
A. Correct.
Q. The brake pads on the bridge mechanism are exposed to

much more forces than the brake pads on a vehicle?
A. Well, again not testifying as a bridge engineer, but my understanding of -- of the way that the -- of the bridge gantry cable system works, the -- the brakes aren't slowing down the -- the section that's moving -- the moveable section. It's the cables and the motors that slow that down. And the -- the brakes lock after that has stopped. So it's not like you're trying to slow down a runaway train with -- with the -- the brake system.
Q. You -- so you don't think there -- brake pads are exposed to more force in a -- in a bridge mechanism than they are in your typical vehicle?
A. Yeah, I -- no, I would agree with that.
Q. All right. Well, that --
A. Yeah.
Q. I'm sorry.
A. Yeah.
Q. That's what I asked.
A. All right.
Q. All right. And --
A. There's different --
Q. -- the brake --
A. -- different -- different type of a force.
Q. When you're driving around the countryside in a

vehicle, the brake pad -- I mean there's a tremendous amount of environmental forces that's spreading any asbestos towards or around in the countryside -- wind, the movement of the vehicle --

A. I don't --

Q. -- all of those things.

A. I don't -- I wouldn't disagree with that.

Q. All right. The brakes pads in the -- in the Carleton Bridge are in an enclosed room.

A. Correct.

Q. So that dust doesn't freely spread around the countryside like it would be in driving around in a postal vehicle. A lot of it's going to be retained in that room.

A. But it's not in the control room.

Q. But it's in the --

A. It's in --

Q. -- the machinery space.

A. Okay.

Q. And someone's got to go down there and clean it up. Isn't that fair to say?

A. My -- my experience of -- of those kinds of spaces is that cleaning is sporadic.

Q. Um hmm.

A. Occupation is sporadic.

Q. Would you --
A. Exposure is -- is sporadic.
Q. To -- to the extent we're talking about exposure to brake pad dust, there would be a lot more exposure on the bridge than there would be driving around in someone's vehicle wouldn't -- wouldn't there be?
A. I don't disagree with that.
Q. All right.
A. My -- my point was exposure in a building that's got a lot of asbestos in it and it's distributed everywhere you go -- I think that's a -- that's a mechanism for continued and long-term given a 24 year period. When you look at that, that's a long-term exposure.
Q. Well, it wasn't really 24 years was it? I mean the -- a lot of that asbestos was out of there long before he left the post office.
A. The -- the last -- well, the last abatement that I have is 2005, so a 20-year --
Q. He got there in the late '80's.
A. Well, that makes that pretty close to 20 years.
Q. Most of the asbestos abatement is in the late 1990's isn't it?
A. The stuff that they're still taking out was in I think 2005.

Q. And you agree most of the documentation is from the late '90's?
A. You're not --
MS. DAVIDSON: Are you referring to abatement --
MR. NOONAN: Yeah.
MS. DAVIDSON: -- documents?
MR. NOONAN: Yeah.
MR. BROADHEAD: Well, the DEP documents are as I said dated 1998, 1999, and 2005 I think.

BY MR. NOONAN:
Q. Most of the asbestos -- I mean you agree or disagree with me -- most of the asbestos abatement documents indicate that that was done in the late 1990's?
A. That's true -- well, I'd have to look at that. I don't -- I can tell you what I can see on the -- on the notification records. What's left in the building, I don't know. How much dust is left in the building, I don't know.
Q. But you keep talking about 24 years. It's not really 24 years.
A. Well, I -- I think I only mentioned 24 years once, but -- so what I'm saying it's a significant -- the longer period than a ten-month period on the Carleton Bridge --

Q. And even if --
A. -- with -- with different materials.
Q. And even if the -- with regard to the latency period -- those last few years -- even if there was asbestos there, that wouldn't play any role in the development of his disease in 2017 would it?
MS. DAVIDSON: Objection --
MR. BROADHEAD: I can't -- I can't --
MS. DAVIDSON: -- foundation and argumentative.
MR. NOONAN: Okay.
MR. BROADHEAD: Yeah.
MR. NOONAN: Okay. Well, I'm not trying --
MR. BROADHEAD: Yeah.
MR. NOONAN: -- to be argumentative.
MR. BROADHEAD: No.
MR. NOONAN: All right.

BY MR. NOONAN:
Q. Are you able to testify about the length of exposure and the likely development of the disease or is that more of a medical question?
A. I think that's more of a medical question. But I think from an industrial hygiene point of view, the longer the exposure -- that's a significant affect.
MR. NOONAN: I don't have any other questions.

MS. DAVIDSON: Nothing further.

ADMINISTRATIVE LAW JUDGE: Is there anything else for the record today?

MR. NOONAN: No.

MS. DAVIDSON: No.

ADMINISTRATIVE LAW JUDGE: All right. We can go off record.

OFF THE RECORD

(Wherefore the above-entitled proceedings were concluded on this date.)

I, Michele Grant, do attest and hereby certify that the preceding is a true transcript of a digital recording of a workers' compensation hearing held in this case on November 27th, 2018.

Michele Grant, Transcriptionist

Word-4-Word Transcription Service

NTC NORTHEAST TEST CONSULTANTS

PROFESSIONAL PROFILE

Stephen R. Broadhead:
Chartered Chemist, Member of the Royal Society of Chemistry, ABIH Certified Industrial Hygienist (CIH) in the Comprehensive Practice of Industrial Hygiene, Certified Microbial Consultant (CMC)
Title: President of NORTHEAST TEST CONSULTANTS

Mr. Broadhead has over forty (40) years of experience in the field of chemical control & corrosion, safety & health, indoor air quality, asbestos/lead related projects, and industrial hygiene through his consulting and power generation career in the United States and the United Kingdom and as president of NORTHEAST TEST CONSULTANTS.

Mr. Broadhead is a Chartered Chemist, Chartered Scientist, Member of the Royal Society of Chemistry, Member of the American Institute of Chemists, Member of the American Industrial Hygiene Association (AIHA), and a Certified Industrial Hygienist (CIH) by the American Board of Industrial Hygiene (ABIH). Mr. Broadhead is also a registered Asbestos Consultant in the State of Maine, as well as a US EPA AHERA Inspector, Design Consultant and Management Planner.

Mr. Broadhead has taken courses for mold certification as a Certified Indoor Environmentalist (CIE) and a Certified Mold Technician (CMT).

Mr. Broadhead has become certified by the American Council for Accredited Certification (ACAC) as a Certified Microbial Consultant (CMC).

Mr. Broadhead has attended various continuing education courses including Indoor Air Quality courses provided by the Harvard Education Resource Center for Occupational Safety and Health, Maine Indoor Air Quality Council, etc.

Mr. Broadhead is an instructor for various training courses provided through NORTHEAST TEST CONSULTANTS ENVIRONMENTAL TRAINING CENTER.

Mr. Broadhead has provided numerous expert witness testimonies for environmental exposure litigation cases, including mold related activities.

(207) 854-3939 www.netest.com

NORTHEAST TEST CONSULTANTS

Steve Broadhead
Consultation and Expert Witness Testimony References

Various Commercial and Industrial Clients	OSHA Compliance Training for Cadmium, Lead, Asbestos, Respiratory Protection, Workplace Safety, etc.
Ken Gray Pierce Atwood One Monument Square Portland ME 04101	Document Review and Consultation
Rich Bayer Robinson, Kriger & McCallum 12 Portland Pier PO Box 568 Portland ME 04112-0568	Expert Testimony & Consultation
Tim Bryant & Jeff Edwards Preti Flaherty One City Center PO Box 9546 Portland ME 04112-9546	Indoor Air Quality Testing / Consultation Expert Testimony
Bruce Hepler, Esq. 75 Pearl St, #214 Portland ME 04101	Indoor Air Quality Testing / Consultation Expert Testimony
John Giffune Verrill & Dana One Portland Square Portland ME 04112	Document Review / Consultation
Robert Kline Kline Law Offices One City Center, 11th Floor PO Box 7859 Portland ME 04112-7859	Consulting for Community Noise Levels
James Poliquin Norman Hanson & Detroy LLC 415 Congress Street PO Box 4600 Portland ME 04112-4600	Professional Services / Consulting